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BRONCHOSPIROGRAPHY

III. THE FUNCTIONAL CAPACITY OF NORMAL LUNGS, SEVERELY DAMAGED LUNGS, LUNGS WITH STRICTLY PARENCHYMAL LESIONS, THORACOPLASTY LUNGS, AND RE-EXPANDED PNEUMOTHORAX LUNGS

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LITERATURE ON BRONCHOSPIROGRAPHY

JACOBAEUS and his associates[6] were the first to describe the method of bronchospirography, using a double-channeled bronchoscope. Already in their first studies they found that the functional capacity of lungs cannot always be predicted by roentgenologic examination. In 1934, Björkman[2] reported studies with this method on normal persons. He found that the vital capacity, as determined by bronchospirometry, is lower than that determined by spirometry, since persons are not able to inhale as deeply with the bronchoscope in place as without it. Therefore, he found the sum of the complementary air from both sides considerably smaller than the spirometric value. The reserve air was often increased; that is, the respiratory mid-position was elevated. Since a patient during bronchoscopy is not in basal condition, his respiratory rate and his oxygen intake are increased as compared to the values found in spirometry. While he found that, in normal persons, ventilation of the right lung is 50 to 60 per cent of the total, he found it in one apparently normal person as high as 67.7 per cent. In the evaluation of the function of the two lungs, oxygen intake appeared as the most constant and important factor. Björkman observed that pleural involvement may considerably decrease the ventilation of the affected side, but the oxygen intake may remain within normal limits in spite of ventilatory impairment. In diseased lungs, on the other hand, oxygen intake is frequently more reduced than ventilation. In this paper, observations are reported on unilateral and bilateral tuberculosis and on cases with pneumothorax and thoracoplasty. These conclusions were restated in a brief communication in 1936 by Jacobaeus' associates, Frenckner and Björkman.[4] Jacobaeus summarized the previous publications in 1936.[7] Bezançon and his co-workers[1]

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studied the function of the two lungs consecutively by the use of a single bronchoscope. They reported that even large pulmonary abscesses do not much impair the function of the lung, while discrete tuberculous lesions may severely limit the pulmonary capacity. Jacobaeus, in a paper read at the annual meeting of the American Association of Thoracic Surgery in 1938,[8] stated that the right lung has approximately 53 to 54 per cent of the total function, and that adhesive pleuritis may greatly reduce function and particularly the expulsive force of cough. Again, he emphasized the fact that the roentgenologic appearance may be misleading as to the functional capacity of the lung, and he cited especially one case with bronchial stenosis in which the roentgenogram showed air-containing lung tissue, but the lung on the stenosed side did not participate in oxygen intake. He drew attention to the fact that oxygen intake becomes impaired earlier than carbon dioxide output. He recommended bronchospirometry as a method to determine indications and contraindications for surgical collapse measures.

In 1939 Gebauer[5] reported a soft-rubber bronchial catheter, constructed for use in bronchospirography. Zavod[12], in 1940, described a similar catheter that he had designed independently of Gebauer. The latter discussed pulmonary volumina and oxygen intake in seven patients, while Leiner, Pinner, and Zavod,[11] working with Zavod's catheter, studied the functional changes brought about by collapse procedures, stressing particularly the compensatory mechanisms that follow collapse. Both Gebauer and Zavod pointed out that bronchospirography with a soft-rubber catheter is a less traumatizing procedure and less disagreeable for the patient than that done with the bronchoscope. The estimation of vital capacity with soft-rubber catheters comes considerably closer to the results with spirometry than that done with the bronchoscope. Whether or not this is true also for other ventilation factors and for oxygen intake remains to be determined.

In two papers[9, 10] that appeared after Jacobaeus' death, he and Bruce studied the "ability of the lungs to substitute for one another." Under normal conditions, either lung is able to assume the total function promptly, as soon as the other lung is functionally eliminated either by bronchial obstruction or by breathing nitrogen. A similar capacity was observed in certain lungs with severe morbid changes.

THE PLACE OF BRONCHOSPIROGRAPHY

The desirability of functional tests for the right and left lung separately needs hardly more justification than separate renal function tests. Since the introduction of bronchospirometry by Jacobaeus in 1932, not many patients have been examined by this method so that its reliability and applicability need still be determined.

From the point of view of physiologic interest, bronchospirography will aid in establishing more definitely the compensatory mechanisms that are called forth in one lung when the other lung is functionally damaged. Spirometric examinations alone can determine the functional capacity and reserve of the patient as a whole, but they cannot measure the degree of functional limitation that disease or treatment has caused in one lung, since the total function is determined by the degree of functional damage *and* the contralateral compensatory function. For the same reason, spirometry cannot measure the degree of compensation or determine its nature.

For practical application, that is, essentially in the attempt to establish contraindications to collapse therapy in single patients, it is, at times, not enough to know the total function, since the participation of left and right lungs may determine whether collapse procedures can be tolerated. A patient with considerably reduced functional reserve is found to have about as much function on the right as on the left; an extensive thoracoplasty is likely to cause respiratory failure; however, if it should be found that the lung considered for thoracoplasty contributes only 10 per cent to the total function, thoracoplasty can probably be done with safety.

All of our studies were done with the soft-rubber catheter described by Zavod. Each patient had previous or subsequent spirometric tests in which the usual kymographic tracings were made while the patient breathed room air and pure oxygen and was in a basal condition and following a brief period of exercise. In regard to these tests we followed closely the methods described by Cournand, Richards, and Darling,[3] who were generous in giving us their personal advice.

In the bronchospirographic tests, the patient always breathed pure oxygen. Exercise tests are hardly possible with the bronchial catheter in place, and they were omitted. In each test the following pulmonary volumina were determined: vital capacity, reserve air, tidal air, and complementary air. In addition, the oxygen intake was measured. An attempt was made to determine the maximum breathing capacity, but it is obvious from our records that in many cases this determination is unreliable, probably because the bronchial catheter becomes easily dislodged under maximal respiratory efforts. In each case, and for each lung separately, the ventilation equivalent was calculated. The ventilation equivalent is the amount of air, in liters, that must be ventilated in order that 100 c.c. of oxygen be resorbed.

In order to make the conditions in spirometry as comparable as possible to those in bronchospirometry, practically each patient was subjected to a second spirometric test in which he had to breathe against a stenosis of about the same magnitude as that presented by the bronchial catheter.

Ideally, the sum of the function of the left lung plus that of the right lung should be equal to the total, as determined by spirometry. This is not the case, save within reasonable limits for vital capacity. In eighty-eight tests, spirometric (with stenosis) and bronchospirometric determinations of vital capacity were done. It was found that the mean deviation of the latter was minus 1.9 per cent of the former. Of all tests, 68 per cent of bronchospirometric estimations were between minus 12 plus 10 per cent of the spirometric values.

Oxygen intake in bronchospirometry is, with few exceptions, higher than in spirometry, with or without stenosis. This is due to the fact that the patient is not under basal condition. There are a certain amount of physical effort associated with the introduction of the catheter, the undetermined effect of local anesthesia, apprehension, some physical discomfort, and the changes in the ventilatory and respiratory mechanism that are caused by the fact that the patient must breathe against a stenosis. Since all these factors, except the last, are in a degree dependent upon vastly different subjective factors, the increment of oxygen intake in bronchospirometry over spirometry varies from one patient to the other.*

The measure of absolute oxygen intake during bronchospirometry is, according to the discussion above, irrelevant. The important factor is the percentage distribution between left and right lung.

The mid-position of the lung is shifted while a patient breathes against a stenosis. The absolute values for reserve and complementary air are therefore distorted in bronchospirography, and no definite conclusion should be based on them. On the other hand, the percentage distribution between left and right is valid and important. The magnitude of the tidal air of one lung in relation to the vital capacity of the same lung is indicative of the amount of ventilatory reserve of this lung. In our tables, therefore, "tidal air in per cent of vital capacity" is frequently shown. This factor appears considerably more significant than the analogous factor for both lungs combined, since a "good" lung will obscure this factor in a "poor" lung. The same holds true for the ventilation equivalent. Since it is the numerical expression of the relation between ventilatory and respiratory functions, its true magnitude is obscured when it is measured as *one* factor relating to one well and one poorly functioning lung.

*To which degree correlation exists (1) between the data from the usual spirometry and those from spirometry during stenosis breathing and (2) between the latter and those from bronchospirometry will be the subject of a later communication.

The legitimate place of bronchospirography in the field of pulmonary function tests can, then, be specified clearly. *Bronchospirography will determine to what extent each lung contributes to the total pulmonary function, as measured by appropriate spirometric studies.* Bronchospirographic data should not be used in an attempt to measure the total function of the lungs. It follows logically that the main field of practical application is collapse therapy when the balance between the two lungs is potentially so severely disturbed.

It might be asked to what extent bronchospirography is an important method in determining contraindications for collapse therapy, particularly for irreversible measures. We are not prepared to give a definite answer to this question. It will depend to a large extent on the possibility of appraising functional capacity from other available data, particularly roentgenologic and clinical. As long as we continue to find that a lung has a much poorer or a much better functional capacity than we expected according to roentgenologic and other evidence, we feel fully justified in continuing routine preoperative tests.

In the present publication we wish to present a few groups of cases according to some definite classifications that seemed to us to be of particular interest.

NORMAL LUNGS

We have no studies on normal persons, save one. But even with a fairly large series of normal persons it seems unlikely that one can arrive at normal standard data, since bronchospirography, as discussed above, introduces certain factors (physical strain, local anesthesia, apprehension, stenosis breathing) whose individual effects are of such differing magnitude that normal values, in the usual sense of the word, could hardly be established.

Our studies were all done on patients with definite pulmonary disease. We have selected thirty-one who had, according to roentgenologic appearance, one practically normal lung, in order to study the bronchospirographic data obtained from such lungs. Neither the lungs nor their function can be considered strictly normal for essentially two reasons. (1) A roentgenogram may not disclose previous lesions which may have damaged the pulmonary function without leaving any roentgenologic evidence. This is especially true for exudative or dry pleuritis that may have caused obliteration of the pleural space. It is true to a lesser extent of parenchymal foci that have resorbed. Moderate degrees of emphysema may not be evident on x-ray examination. (2) Since all patients examined had active disease in one lung, some treated by collapse measures, the apparently "normal" lung will in many cases have undergone certain compensatory changes which may be apparent in its function but not in its roentgenologic appearance.

Normality is indivisible; if one lung is functionally not normal, the other lung is most likely not normal in its function. The data which we present must be understood within the limitations mentioned and not as really normal findings. They are, however, representative of the

so-called good contralateral lung in patients with pulmonary tuberculosis who may come under consideration for medical or surgical collapse.

TABLE I

BRONCHOSPIROGRAPHIC DATA OF ROENTGENOLOGICALLY "NORMAL" LEFT LUNGS

PATIENT NO.	CONTRALATERAL LESION	O_2 INTAKE OF LEFT LUNG IN PER CENT OF TOTAL	VENTILATION EQUIVALENT (LEFT LUNG)	VITAL CAPACITY OF LEFT LUNG IN PER CENT OF COMPUTED TOTAL VITAL CAPACITY	TIDAL AIR OF LEFT LUNG IN PER CENT OF TOTAL TIDAL AIR	TIDAL AIR OF LEFT LUNG IN PER CENT OF VITAL CAPACITY OF LEFT LUNG	RESERVE AIR OF LEFT LUNG IN PER CENT OF VITAL CAPACITY OF LEFT LUNG	COMPLEMENTARY AIR OF LEFT LUNG IN PER CENT OF VITAL CAPACITY OF LEFT LUNG
50	F. a., residual empyema	78	1.2	35	47	19	30	51
	Upper stage thoracoplasty	76	1.7	38	66	18	33	49
51	Mod., phrenic	72	2.1	40	68	49	20	31
	7-rib thoracoplasty	89	2.2	36	75	30	30	40
54*	Min.	56	3.1	37	53	16	34	50
57*	Normal	49	3.6	44	54	19	34	47
59*	Mod.	60	3.3	31	69	9	52	39
	2-stage 6-rib thoracoplasty	68	2.5	26	52	21	15	64
60	Mod., phrenic	74	2.3	30	62	18		
72*	Mod.	43	2.2	41	51	11		
73	5-rib thoracoplasty	66	2.5	35	53	29	38	33
74	Fibrothorax	82	2.1	55	55	15	37	48
79*	Min.	52	2.3		51			
	5-rib thoracoplasty	54	2.0	38	52	18	34	48
87	40% pneumothorax collapse	64	2.3	48	60	19	36	45
	Lysis 80% collapse	76	1.8	39	67	26	16	58
127	Bronchial carcinoma	75	2.5	29	61	25	33	42
140	Mod.	61	3.5	38	53	22	27	51
142	F. a.	58	3.9	25	57	31	13	56
150*	Mod.	42	4.4	47	51	19	15	66
158*	Min. bilateral	53	2.2	43	36	20	47	33
215	F. a.	58	2.1	57	53	11	32	57

*Lungs closest to completely normal condition. The means, maxima, and minima pertaining to these seven lungs are presented in Table III.

Since normal standards in absolute figures are not available, it is necessary to present bronchospirographic data not in their theoretically assumed relation to spirometric data but in percentages of total function. As mentioned, in practically all of our patients spirometric readings were taken while they were breathing against a stenosis of the

same magnitude as is presented by the bronchial catheter. Some of our data suggest that there is a wide individual variation in the response to stenosis breathing, and we have no evidence to assume that these variations are dependent upon particular pathologic changes.

TABLE II

BRONCHOSPIROGRAPHIC DATA OF ROENTGENOLOGICALLY "NORMAL" RIGHT LUNGS

PATIENT NO.	CONTRALATERAL LESION	O_2 INTAKE OF RIGHT LUNG IN PER CENT OF TOTAL	VENTILATION EQUIVALENT, RIGHT LUNG	VITAL CAPACITY OF RIGHT LUNG IN PER CENT OF TOTAL COMPUTED VITAL CAPACITY	TIDAL AIR OF RIGHT LUNG IN PER CENT OF TOTAL TIDAL AIR	TIDAL AIR OF RIGHT LUNG IN PER CENT OF VITAL CAPACITY OF RIGHT LUNG	RESERVE AIR OF RIGHT LUNG IN PER CENT OF VITAL CAPACITY OF RIGHT LUNG	COMPLEMENTARY AIR OF RIGHT LUNG IN PER CENT OF VITAL CAPACITY OF RIGHT LUNG
25	Complete collapse, empyema	91	1.5	32	86	22	21	57
44	40% pneumothorax collapse	79	2.2	43	66	14	39	47
55	6-rib thoracoplasty	67	2.7	33	53	33	21	46
56	Fibrothorax, pneumothorax effusion, endobronchial tuberculosis	80	2.6	39	59	18	31	51
57*	Normal	51	2.9	36	46	19	35	46
58	5-rib thoracoplasty	74	3.6	37	64	23	10	67
	Same, revision	73	2.5	28	46	15	21	64
62	7-rib thoracoplasty	85	2.1	28	73	20	35	45
151	Mod., obliterative pleuritis	76	1.8	44	50	13	50	37
156	Obliterative pleuritis, min.	76	2.4	39	52	17	27	56
157	Mod. pneumothorax obliteration	77	2.1	38	61	27	42	31
186*	Min.	66	3.1	45	57	11	29	60
192	Min.	72	2.6	38	50	21	16	63
241	Diaphragmatic adhesions, f. a.	76	2.5	43	58	22	42	36
247	Mod., pleural symphysis	88	2.2	47	79	16	40	44

*Lungs closest to completely normal conditions.

On the thirty-one patients selected, thirty-seven separate tests are reported (Tables I and II) which include a few repeated tests following contralateral collapse therapy; twenty-two are of the left and fifteen of the right lung. It is obvious, at once, that a number of these "apparently normal lungs" are not normal functionally, as shown by the large share that they have in the total oxygen intake. It is prob-

ably justifiable to exclude from consideration, solely on the basis of this criterion, all left lungs with an oxygen intake whose percentage of the total is more than 60. Theoretically the left lung, under completely normal conditions, should contribute, according to Björkman,[2] 46 to 47 per cent to the total oxygen intake. By widening the margin up to 60 per cent, we probably make allowance for inherent technical sources of error and for certain deviations from normal that have no great practical importance. The lungs so omitted from Table I and the reasons for their elevated oxygen intake, which are to be found in the contralateral lung, are given in Table III.

TABLE III

PATIENT NO.	CONTRALATERAL LESIONS
50	Far-advanced cavitary disease, empyema, followed by 2-stage thoracoplasty
51	Permanent diaphragmatic paralysis, moderately advanced disease, followed by 2-stage thoracoplasty
59	Second test: 2-stage thoracoplasty
60	Phrenic paralysis and moderately advanced disease
74	Fibrothorax, following pneumothorax and effusion
73	5-rib thoracoplasty, pleural obliteration
87	40% pneumothorax collapse Second test: 80% collapse following pneumonolysis
140	Moderately advanced disease; i.e., *no definite reason*
127	Bronchogenic carcinoma with occlusion of upper lobe bronchus

Table III accounts satisfactorily for the increased oxygen intake in all but one case. However, it is interesting to see that one case (Case 79) maintained a "normal" oxygen intake following a five-rib thoracoplasty on the contralateral side and that two cases (Cases 142 and 215) with far-advanced parenchymal disease in the contralateral lung fell, according to the criterion of oxygen intake, in the "normal" group. Furthermore, it is probably of considerable interest that none of the left lungs that were selected as normal according to roentgenologic evidence had significantly less than the theoretically normal oxygen intake of 46 to 47 per cent.

If, on similar consideration, all those right lungs (Table II) were excluded from the "normal" group that have an oxygen intake of more than 70 per cent, only three such lungs would be left out of a total (including one repeated test) of fifteen. Björkman found in normal right lungs a percentage of oxygen intake up to more than 60 per cent. These three lungs are those of (1) Case 57, a normal volunteer, (2) Case 55, a patient with a contralateral six-rib thoracoplasty, and (3) Case 186, a patient with minimal contralateral disease.

In the other cases, the increase in oxygen consumption of the right lung appears fully explained by the conditions of the left lung: extensive thoracoplasties in two patients (Cases 62, 58) and three tests; obliteration of the pleural space with or without preceding pneumothorax and effusion in five patients (Cases 56, 151, 156, 157, 247); existing pneumo-

thorax in one patient (Case 44), extensive pyopneumothorax in one patient (Case 25) and far-advanced disease with an elevated and tented diaphragm in one (Case 241). Only one case (Case 192) remains unexplained. This patient had only minimal, fibronodular disease in the left upper lobe. In a later paragraph it will be shown that pleural obliteration per se may reduce the respiratory functions to a great degree. It is then probably safe to assume that in Case 192 the left pleural space is obliterated.

With the available material it seems impossible to reach any definite conclusions as to normal standards for bronchospirographic data, especially since there is no strict parallelism between lungs that cannot be considered normal because of the extent of contralateral involvement and those that are obviously not normal because they show evidence of compensatory hyperfunction. When all lungs which are apparently not normal, according to either the clinical or the functional criteria mentioned, are omitted, there remain only seven left lungs (Cases 59, 72, 79, 150, 158, 57, and 54) and two right lungs (Cases 57 and 186) which may be considered approximately normal. The means, maxima, and minima of these seven left lungs are presented in Table IV.

TABLE IV

MEANS, MAXIMA, AND MINIMA OF SEVEN LEFT LUNGS (TABLE I) CLOSEST TO NORMAL CONDITIONS

	O_2 INTAKE OF LEFT LUNG IN PER CENT OF TOTAL	VENTILATION EQUIVALENT	VITAL CAPACITY OF LEFT LUNG IN PER CENT OF COMPUTED TOTAL VITAL CAPACITY*	TIDAL AIR OF LEFT LUNG IN PER CENT OF TOTAL TIDAL AIR	TIDAL AIR OF LEFT LUNG IN PER CENT VITAL CAPACITY OF LEFT LUNG	RESERVE AIR OF LEFT LUNG IN PER CENT OF VITAL CAPACITY OF LEFT LUNG	COMPLEMENTARY AIR OF LEFT LUNG IN PER CENT OF VITAL CAPACITY OF LEFT LUNG
Mean	51	3.0	41	54	16	36	47
Maximum	60	4.4	47	69	20	47	66
Minimum	42	2.2	31	36	9	15	33

*The mean, maximum, and minimum of vital capacity in per cent of total vital capacity are 54, 65, and 46 respectively.

In all, but one, of these seven lungs that are apparently closest to normal, the respiration (oxygen intake) is a greater percentage of the total respiration than the vital capacity is of the total vital capacity. It cannot be decided whether this means that respiration is increased with a corresponding increase in vital capacity, or whether it is an expression of the increase in oxygen intake because of the nonbasal conditions of the patients. The tidal air is, on the average, 16 per cent of the vital capacity, which is close to the spirometrically normal value, as derived from the literature.

The reserve air, spirometrically determined, is supposed to be about 25 per cent of the vital capacity, and the complementary air, about

62 per cent. Our bronchospirographic data, with the means of reserve air higher than the theoretical normal and the means of the complementary air lower, indicate that, probably due to stenosis, the midposition of the lung is usually shifted toward the inspiratory position. In a later communication we shall discuss in detail the changes in ventilation caused by stenosis breathing.

It should be pointed out in conclusion that roentgenologically normal lungs have in our experience never taken a distinctly smaller than normal part in the total function under the conditions of bronchospirographic tests which is somewhere between the basal status and exercise. However, roentgenologic appearance will not indicate to what degree such a lung has already exhausted part of its reserve capacity.

SEVERELY DAMAGED LUNGS

It seemed of interest to find out what type of lesions and what type of collapse procedures are most likely to inflict particularly severe functional damage. For this purpose we have selected all patients who in one lung absorbed only 25 per cent or less of the total oxygen intake. This is an arbitrary choice, but it insures the selection of severely impaired lungs.

Of a total of ninety-eight patients with 115 tests who were submitted to bronchospirography to date, nineteen fell in this group; in fourteen, the left lung was gravely impaired, and in five, the right. The bronchospirographic data that seem pertinent are summarized in Tables V and VI. The percentage of oxygen consumption of the damaged lung is far below the upper limit allowed for by the selection; in one-half of the patients it is below 15 per cent. It will be seen that in all severely damaged lungs, the ventilation equivalent is high, in most of them above 5. Normal values (2.3, 1.9) were found in only two patients. In one (Case 25), chronic empyema has practically immobilized the left hemithorax; in the other (Case 46) the high ventilation equivalent of 10.3 was reduced by thoracoplasty which apparently closed an apical cavity and eliminated ventilation of nonrespiring lung tissue. It would be expected that the ventilation equivalent is *one* significant indicator of the impaired respiratory function, being an index of the efficiency of ventilatory function in terms of oxygen intake. However, in spirometric studies poor pulmonary function is frequently not associated with a high ventilation equivalent. This is, at least in part, explained by the fact, shown in Table V, that the ventilation equivalent of the two lungs, as measured by spirometry, obscures frequently the high ventilation equivalent for *one* lung. A ventilation equivalent much lower (and frequently normal) than that of a single lung is strikingly demonstrated in Cases 46, 56, 69, 61, 231, 154, and 171. The high ventilation equivalent is proof that respiration is more severely damaged than ventilation. The vital capacity is much less reduced than the oxygen intake, indicating that the ventilatory reserve

TABLE V

BRONCHOSPIROGRAPHIC AND CLINICAL DATA OF THIRTEEN SEVERELY DAMAGED LEFT LUNGS

PATIENT NO.	CLINICAL DATA	O_2 INTAKE OF LEFT LUNG IN PER CENT OF TOTAL	VENTILATION EQUIVALENT			VITAL CAPACITY OF LEFT LUNG IN PER CENT OF TOTAL	TIDAL AIR OF LEFT LUNG IN PER CENT OF TOTAL TIDAL AIR	TIDAL AIR OF LEFT LUNG IN PER CENT OF VITAL CAPACITY OF LEFT LUNG	RESERVE AIR OF LEFT LUNG IN PER CENT OF TOTAL RESERVE AIR	COMPLEMENTARY AIR OF LEFT LUNG IN PER CENT OF TOTAL COMPLEMENTARY AIR
			BRONCHOSPIROMETRY		SPIROMETRY					
			L	R						
25	L: pneumothorax 4 years, pyopneumothorax 2 years 90 per cent collapse with thick pleura, left chest shrunken	9	2.3	1.5	2.4	14	14	22	23	12
44	L: pneumothorax 4 months, lysis, no fluid	21	4.4	2.2	2.9	23	34	24	6	30
46	L: pneumothorax 6 years, re-expanded 1 year ago. Fibrothorax, apical cavity	9	10.3	2.3	2.2	25	28	25	7	19
	Following 2-stage 5-rib thoracoplasty	9	1.9	1.4	2.3	17	12	10	9	20
56	L: pneumothorax 19 months, obliterated 12 months ago. Fibrothorax with marked retraction	20	7.4	2.6	2.2	20	41	48	11	16
61	L: pneumothorax for 1 year 6 years ago, 5 months ago 2-stage 5-rib thoracoplasty, before thoracoplasty fibrothorax	20	6.8	2.1	3.3	25	45	41	17	23

62	L: pneumothorax impossible, phrenicectomy 3 years ago, 2-stage 7-rib thoracoplasty 16 months ago. Fair collapse upper half	15	4.6	2.1	2.9	20	27	31	17	18
69	L: pneumothorax induced 7 years ago, abandoned after 4 years (pneumothorax, right, 2 years). Fibrothorax with retraction. R: apical cavity	12	16.4	2.2	1.9	59	50	28	53	73
102	L: pneumothorax induced 30 months ago, obliterated 24 months later. Fibrothorax. R: exudative lesion r. u. l. with 3 cm. cavity	20	4.9	2.2	2.7	16	34	37	10	14
151	L: pleuritis 17 years ago, pneumothorax impossible, 1 year ago. 2-stage 6-rib thoraplasty 5 months ago. Good upper collapse	24	6.1	2.5	5.1	29	43	32	15	29
227	L: pneumothorax for 29 months abandoned 3 years ago, massive effusion 2 years ago, replaced by air. Hydropneumothorax	14	5.3	1.4	4.8	23	40	39	21	15
231	Bilateral dissemination of nodular lesions in upper third, somewhat more on left; pneumothorax, left impossible	22	7.3	2.7	2.0	26	44	54	7	26
247	L: effusion 8 years ago, later pneumothorax impossible. Fibrocavitary lesion between apex and 2 anterior ribs	12	4.4	2.2	3.5	21	21	16	22	21
253	L: pneumothorax for 2 years, discontinued 3 years ago. Very thick pleura and basal effusion, diffuse parenchymal fibrosis	18	4.2	2.1	3.6	20	30	50	13	18

TABLE VI

BRONCHOSPIROGRAPHIC AND CLINICAL DATA OF FIVE SEVERELY DAMAGED RIGHT LUNGS

PATIENT NO.	CLINICAL DATA	O_2 INTAKE OF RIGHT LUNG IN PER CENT OF TOTAL	VENTILATION EQUIVALENT			VITAL CAPACITY OF RIGHT LUNG IN PER CENT OF TOTAL	TIDAL AIR OF RIGHT LUNG IN PER CENT OF TOTAL TIDAL AIR	TIDAL AIR OF RIGHT LUNG IN PER CENT OF VITAL CAPACITY OF RIGHT LUNG	RESERVE AIR OF RIGHT LUNG IN PER CENT OF TOTAL RESERVE AIR	COMPLEMENTARY AIR OF RIGHT LUNG IN PER CENT OF TOTAL COMPLEMENTARY AIR
			BRONCHOSPIROMETRY		SPIROMETRY					
			L	R						
28	R: pneumothorax 3 years, endobronchial tuberculosis, lysis 7 months ago, collapse 90 per cent, no fluid	9			2.3					
51	R: exudative pleuritis 13 years ago, phrenicectomy 2 years ago, 2-stage 7-rib thoracoplasty	11	2.2	6.3	2.6	13	32	67	5	8
74	R: pneumothorax 4 years with clear effusion; re-expanded 10 months ago. Fibrothorax with marked retraction	18	2.1	8.2	2.8	15	45	65	8	6
154	R: phrenicectomy 7 years ago, pneumothorax 8 months, abandoned 5 years ago. R. diaphragm elevated, extensive fibrocaseous lesion	19	2.2	5.8	3.3		39			
171	R: pneumothorax induced 7 months ago, lysis 4 months ago, tuberculous empyema and bronchopleural fistula, thoracotomy; thick pleura	4	1.8	6.3	2.9	5	12	52	6	6

is frequently much less impaired than the respiratory function. Both complementary and reserve air are reduced, the former roughly in the same proportion as the vital capacity, while the reserve air shows disproportionately large reduction. In other words, the mid-position of these severely damaged lungs is usually closer to the expiratory phase than in normal lungs; the reverse is true in emphysema. One noticeable exception to this rule is Case 69, a woman who has had bilateral successive pneumothorax. The tidal air in percentage of the total tidal air is far less reduced than any of the other ventilatory factors shown; in a number of cases it is approximately normal (Cases 61, 69, 151, 231, and 74), although all of these cases with the exception of Case 69 show extensive impairment of oxygen intake. However, in all cases except Case 46 (following thoracoplasty) the tidal air is a much larger than normal part of the available vital capacity, showing that the ventilatory reserve is severely reduced.

TABLE VII

SEVERELY DAMAGED LUNGS; MEANS, MAXIMA AND MINIMA FROM TABLES V AND VI

	O_2 INTAKE IN PER CENT OF TOTAL	VENTILATION EQUIVALENT			VITAL CAPACITY OF DAMAGED LUNG IN PER CENT OF TOTAL VITAL CAPACITY	TIDAL AIR IN PER CENT OF TOTAL TIDAL AIR	TIDAL AIR IN PER CENT OF VITAL CAPACITY OF SAME LUNG	RESERVE AIR OF DAMAGED LUNG IN PER CENT OF TOTAL RESERVE AIR	COMPLEMENTARY AIR OF DAMAGED LUNG IN PER CENT OF TOTAL COMPLEMENTARY AIR
		BRONCHOSPIROMETRY		SPIROMETRY					
		DAMAGED LUNG	CONTRALATERAL LUNG						
No. of experiments	19	18		19	17	18	17	17	17
Mean	15	6.3	2.1	2.9	22	33	38	15	21
Maximum	24	16.4	4.4	5.1	59	50	67	53	73
Minimum	4	1.9	1.4	1.9	5	12	10	5	6

The data discussed are summarized in terms of means, maxima, and minima in Table VII. It is shown clearly in this table that oxygen intake (respiration) is more severely impaired than vital capacity, that the ventilation equivalent, the numerical expression of the relation between ventilatory and respiratory functions, is high, although this is rather obscured when measured for the two lungs together, and that there is a tendency for these lungs to be in relative expiratory position, as indicated by the values for reserve and complementary air. The tidal air constitutes a very large percentage (38) of the vital capacity. A study of the individual cases shows these changes in a more emphatic manner in the majority of instances.

From the clinical data it is apparent that the outstanding factor responsible for the particularly poor function is concerned primarily with the pleura. Each patient has had pneumothorax, followed by fibrothorax, pleural obliteration, or unsuccessful pneumothorax attempts or

pleuritis, in other words, all conditions that involve fibrotic and/or obliterative pleuritis. The actual extent of active lesions plays an entirely subordinate role. This is particularly clearly brought out by the fact that more than half of these patients had at the time of bronchospirography active lesions of various extent in the contralateral well functioning lung.

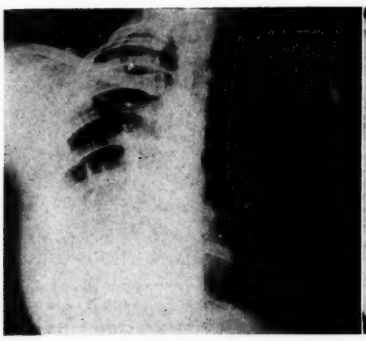

Fig. 1.

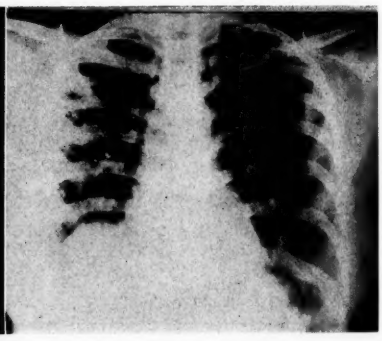

Fig. 2.

Fig. 1.—Roentgenogram of Case 74 in Table V. Fibrothorax, right, following pneumothorax with clear intrapleural effusion.

Fig. 2.—Roentgenogram of Case 154 in Table V. Diaphragmatic paralysis, right, obliterated pleural space, fibrocaseous lesions.

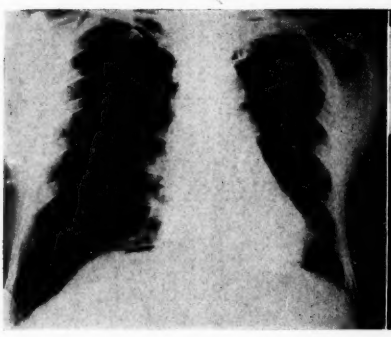

Fig. 3.

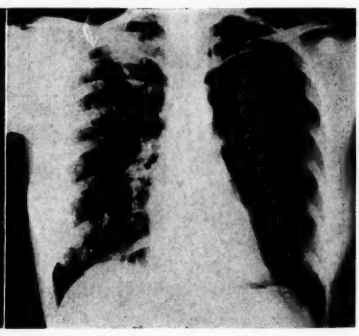

Fig. 4.

Fig. 3.—Roentgenogram of Case 247 in Table IV. Small fibrocavitary lesion in left upper lobe and slight shift of mediastinal structure to left. Pleural space on left is obliterated.

Fig. 4.—Roentgenogram of Case 158 in Table VII. Moderately advanced, infiltrative tuberculosis in right upper lung field, without cavities.

In this series, there are three patients with phrenicectomy. It is our impression that a paralyzed diaphragm (with or without thoracoplasty) contributes considerably to impairment of function, but larger series are necessary, of course, to prove this point. Similar observations concerning the effect of diaphragmatic paralysis on pulmonary function are reported by Jacobaeus[8] and Cournand and Richards.[13]

In Figs. 1 to 3 the roentgenograms of three representative cases with severely damaged lungs are shown. They are Cases 74, 154, and 247, respectively, whose bronchospirographic data are summarized in Tables V and VI. While in the first case profound functional damage may be expected according to the roentgenologic appearance of fibrothorax, this is less definitely the case in the second patient (Case 154), although the elevated diaphragm (that is fixed) should arouse grave suspicion. In the third case (Case 247) there is but minimal parenchymal disease in the left lung; there is a slight shift of the mediastinal structures to the left, but it seems impossible to predict from the roentgenologic appearance that the left lung has such a poor function in comparison to the right, particularly since the right lung is obviously emphysematous. The unreliability of the roentgenogram as an indicator of pulmonary function becomes particularly obvious when Figs. 1, 2, and 3 are compared with Figs. 4, 5, and 6, showing lungs whose function is but little reduced.

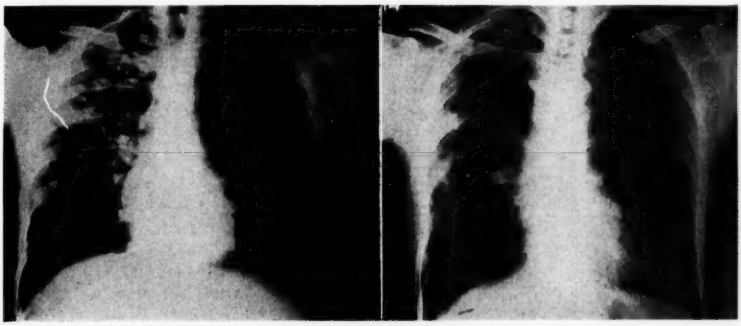

Fig. 5. Fig. 6.

Fig. 5.—Roentgenogram of Case 193 in Table VII. Far-advanced, mainly exudative tuberculosis of right upper lung field, with multiple cavities.

Fig. 6.—Roentgenogram of Case 219 in Table VII. Far-advanced tuberculosis in right mid-lung field with large cavity.

LUNGS WITH STRICTLY PARENCHYMAL LESIONS

In order to compare the functional damage done by active parenchymal disease with that brought about by extensive pleural involvement, we have selected eleven patients in whom one lung showed active parenchymal lesions but no evidence of pleural involvement according to history, clinical and roentgenologic findings (Tables VIII and IX). The mean oxygen consumption in these lungs is 42 per cent, as compared to 15 per cent in the former group, a nearly normal value. The mean ventilation equivalent is 3.9 as compared to 6.3, and, while in the former group of lungs the maximum was 16.4, it is here only 6.7. The vital capacity of these lungs in percentage of the total vital capacity is practically normal with a mean of 50 per cent, in spite of the fact

TABLE VIII

BRONCHOSPIROGRAPHIC AND CLINICAL DATA OF LUNGS WITH STRICTLY PARENCHYMAL LESIONS

PATIENT NO.	STATUS OF SIDE REPORTED	SIDE	O_2 INTAKE IN PER CENT OF TOTAL	VENTILATION EQUIVALENT		VITAL CAPACITY IN PER CENT OF TOTAL	TIDAL AIR IN PER CENT OF TOTAL TIDAL AIR	TIDAL AIR IN PER CENT OF VITAL CAPACITY	RESERVE AIR IN PER CENT OF TOTAL RESERVE AIR	COMPLEMENTARY AIR IN PER CENT OF TOTAL COMPLEMENTARY AIR
				BRONCHO-SPIROM-ETRY	SPIROM-ETRY					
118	Fibrocavitary lesion	R		2.8	2.7	48		8	45	55
136	Fibrocavitary lesion	L	31	3.3	2.3	37	41	32	36	35
140	Fibrosis apex to third anterior rib	R	39	4.7	2.3	51	47	19	45	55
150	F. a., multiple small cavities	R	58	3.1	1.9	51	49	17	56	51
158	Mod., no cavities	R	47	4.4	2.2	54	64	31	57	36
186	Min., fibrot. small cavities?	L	34	4.7	2.9	50	43	15	0	58
192	Min. scattered infiltrations u. l.	L	28	6.7	3.9	61	50	14	50	65
193	F. a., mainly exudative	R	44	4.0	2.5	50	57	17	49	48
219	F. a., exudative with cavities	R	59	2.5	3.0	59	54	13	65	57
249	Scattered fibrocavitary lesions	L	44	3.7	3.8	44	48	92*	37	2
251	Shrunken, fibrotic, u. l.	R	38	3.5	2.3	41	45	21	47	39

*This patient is a hypomaniac; he had 4 respirations per minute, which explains the high percentage of tidal air.

that, in most cases, the contralateral lung is not significantly diseased. Reserve and complementary air do not show any striking deviation from those in our "normal" group.

The differences between the "pleural group" and the "parenchymal group" are striking enough to deserve serious attention. In many instances, parenchymal lesions are much more easily diagnosed and demonstrated than pleural involvement. It is, therefore, of great practical importance in making the indication for collapse therapy to realize that frequently pleural involvement leads to severer functional damage than do much more obvious parenchymal lesions.

TABLE IX

STRICTLY PARENCHYMAL LESIONS, MEANS, MAXIMA, AND MINIMA FROM TABLE VIII

	O_2 INTAKE IN PER CENT OF TOTAL	VENTILATION EQUIVALENT		VITAL CAPACITY IN PER CENT OF TOTAL	TIDAL AIR IN PER CENT OF TOTAL TIDAL AIR	TIDAL AIR IN PER CENT OF VITAL CAPACITY	RESERVE AIR IN PER CENT OF TOTAL RESERVE AIR	COMPLEMENTARY AIR IN PER CENT OF TOTAL COMPLEMENTARY AIR
		BRONCHO-SPIROM-ETRY	SPIROM-ETRY					
No.	10	11	11	11	10	10*	11	11
Mean	42	3.9	2.7	50	50	19	44	46
Maximum	59	6.7	3.9	61	64	32	65	65
Minimum	28	2.5	1.9	37	41	8	0	2

*In this column, Case 249 is omitted because this patient had only 4 respirations per minute.

Figs. 4 to 6 are the roentgenograms of Cases 158, 193, and 219 in Table VIII. In spite of rather extensive lesions, their oxygen intake is 47, 44, and 59 per cent, respectively, of the total oxygen intake, which, even for right lungs, are only relatively slight reductions. The last case, in fact, shows a normal percentage of oxygen intake.

THORACOPLASTY LUNGS

In Table X we present bronchospirographic data of twelve tests in eleven patients who have undergone thoracoplasty and who do not show any evidence of active disease on the operated side. The bronchospirographic data concern only the operated side. A few facts that apply to all or most of these cases should be emphasized.

1. With the exception of two cases with seven-rib thoracoplasties, preceded by phrenicectomy, all these partly collapsed lungs participate to a considerable degree in the respiratory function, as measured by their percentage oxygen intake; the four cases with uncomplicated five-rib thoracoplasty to the extent of an average 38 per cent and the three cases with uncomplicated six-rib thoracoplasty to the extent of 29 per cent. This is a strong argument, from the point of view of function, for limited, selective thoracoplasties whenever possible.

TABLE X

BRONCHOSPIROGRAPHIC AND CLINICAL DATA OF THORACOPLASTY LUNGS

PATIENT NO.	TREATMENT AND PRESENT STATUS		O_2 INTAKE IN PER CENT OF TOTAL	VENTILATION EQUIVALENT	VITAL CAPACITY OF OPERATED LUNG IN PER CENT OF TOTAL	TIDAL AIR OF OPERATED LUNG IN PER CENT OF TOTAL TIDAL AIR	TIDAL AIR IN PER CENT OF VITAL CAPACITY OF OPERATED SIDE	RESERVE AIR OF OPERATED LUNG IN PER CENT OF TOTAL RESERVE AIR	COMPLEMENTARY AIR OF OPERATED LUNG IN PER CENT OF TOTAL COMPLEMENTARY AIR
	RIGHT LUNG	LEFT LUNG							
73	1-stage 5-rib thoracoplasty	Clear	34	4.3	34	47	49	23	31
79	2-stage 5-rib thoracoplasty	Clear	46	2.1	43	48	22	45	39
152	2-stage 5-rib thoracoplasty	Min. fibrocalcification	42	3.6	40	50	28	33	44
61	Min. fibrosis in apex	2-stage 5-rib thoracoplasty	30	6.8	25	45	41	17	23
58	Clear	2-stage 5-rib thoracoplasty preceded by pneumothorax and phrenic crush	26	5.8	42	36	18	62	38
		Id. revision operation	28	8.0	57	78	14	52	59
59	2-stage 6-rib thoracoplasty	Clear	30	4.6	31	48	42	25	24
151	Clear	2-stage 6-rib thoracoplasty	23	6.1	57	43	32	15	29
137	Dissem. fibrocalcific lesions	2-stage 6-rib thoracoplasty			27	27	14	20	31
55	Clear	2-stage 6-rib thoracoplasty	33	4.8	37	47	49	21	35
62	Clear	2-stage 7-rib thoracoplasty preceded by phrenicectomy	15	4.6	20	27	31	17	18
51	2-stage 7-rib thoracoplasty preceded by pleurisy and phrenicectomy	Clear	11	6.3	13	25	66	5	8

2. With the exception of two cases (Cases 58 and 151) the vital capacity of the operated side is decreased practically to the same degree as the oxygen intake. The difference in the limitation between oxygen intake and ventilated volume, as expressed by the ventilation equivalent, is great enough so that, in the majority of cases, the operated side must ventilate from two to three times the normal volume per unit oxygen absorbed.

3. In the majority of cases the tidal air is a high percentage of the vital capacity of the same side. In the apparently normal seven lungs that we presented the average tidal air was 16 per cent of the vital capacity. In these thoracoplasties, it is 34 per cent. This must be understood as an expression of the limitation of ventilatory reserve that these lungs have suffered.

4. With the large portion that the tidal air occupies of vital capacity, both reserve and complementary air are relatively small, but there is no definite tendency toward either the expiratory or inspiratory position of the hemithorax in terms of vital capacity. Residual air measurements were not done.

TABLE XI

THORACOPLASTY LUNGS; MEANS, MAXIMA, AND MINIMA FROM TABLE X

	O_2 INTAKE IN PER CENT OF TOTAL	VENTILATION EQUIVALENT	VITAL CAPACITY OF OPERATED LUNG IN PER CENT OF TOTAL	TIDAL AIR OF OPERATED LUNG IN PER CENT OF TOTAL TIDAL AIR	TIDAL AIR IN PER CENT OF VITAL CAPACITY OF OPERATED SIDE	RESERVE AIR OF OPERATED LUNG IN PER CENT OF TOTAL RESERVE AIR	COMPLEMENTARY AIR OF OPERATED LUNG IN PER CENT OF TOTAL COMPLEMENTARY AIR
No. of experiments	9	9	10	10	10	10	10
Mean	32	5.1	39	47	31	31	35
Maximum	46	8.0	57	78	49	62	59
Minimum	23	2.1	25	27	14	15	23

Cases 51 and 62 are omitted from these calculations because of paralyzed diaphragm.

In Table XI, are summarized the means, maxima and minima, according to Table X. However, Cases 51 and 62 are omitted because they each had a paralyzed diaphragm and, therefore, changes in pulmonary function, not attributable to thoracoplasty per se. These two cases have by far the lowest percentage of oxygen intake, vital capacity and complementary air of any thoracoplasty lungs studied. The means for the remaining ten tests show a moderately decreased percentage of oxygen intake, a high ventilation equivalent, and a vital capacity disproportionately large in comparison with the oxygen intake. These lungs contribute their approximately normal share to the total ventilation (about normal vital capacity and tidal air in percentage of total), but the tidal air constitutes a large percentage (31) of the vital capacity.

RE-EXPANDED PNEUMOTHORAX LUNGS

The term "re-expanded lung" following pneumothorax treatment is so firmly rooted in medical usage that its replacement seems hopeless, although, in many cases, it presupposes a condition which has only incompletely developed. The air in the pneumothorax space has been resorbed completely, but the lung, probably in the majority of patients, has not re-expanded to the pretreatment volume. Instead, mediastinal shift, contralateral emphysema, elevated diaphragm, retracted chest wall, and thickened pleura combine to substitute for full re-expansion. Often, this is clearly seen on inspection and on roentgenograms. What this incomplete re-expansion and the parenchymal changes in the lung mean in terms of function cannot be definitely deduced from spirometric readings, since compensatory mechanisms of the contralateral lung obscure the actual loss of function. Bronchospirographic studies supplement the findings of spirometric observations.

In Table XII we have summarized the bronchospirographic data of fourteen lungs treated by pneumothorax and followed by complete resorption of pneumothorax in twelve patients. In this table are included only such "pneumothorax lungs" that, following complete absorption of the intrapleural air, do not present any evidence of active disease. The contralateral side, on the other hand, may show various degrees of active involvement.

It is evident that no general conclusions regarding so-called re-expanded lungs may be drawn from these data, since no trend and no general and common characteristics can be discerned. The re-expanded lung contributes anywhere from 12 to 91 per cent to the total oxygen intake. It is suggestive that the extent of the contralateral functional damage is a decisive factor in determining the percentage intake of the re-expanded side; this seems to hold particularly for the lungs with a high percentage of oxygen intake. It is remarkable to what a high degree a re-expanded lung can function as far as oxygen intake is concerned. How regularly this may be expected to occur must be determined from more data. The roentgenologic appearance of "fibrothorax" seems to indicate rather regularly a severely damaged lung, as shown by Cases 69, 74, 102, and 153 with percentages of oxygen intake of 12, 18, 20, and 23, respectively. Aside from this fairly definite roentgenologic entity, we must conclude from these and a good many other observations that the roentgenologic appearance of a lung is an unreliable indicator of its functional capacity. We have rejected a number of patients for irreversible collapse procedures, because the bronchospirographic data showed an unexpectedly poor function of the seemingly better lung.

It will be noted from Table XII, that there is a close association between poor oxygen and high ventilation equivalent, while a similar parallelism between oxygen intake and vital capacity does not exist (for example, Cases 69 and 235). In all poorly oxygenating lungs the tidal

TABLE XII

BRONCHOSPIROGRAPHIC AND CLINICAL DATA OF RE-EXPANDED PNEUMOTHORAX LUNGS

PATIENT NO.	SIDE	DURATION IN MONTHS	PNEUMOTHORAX			PRESENT STATUS	O_2 INTAKE IN PER CENT OF TOTAL	VENTILATION EQUIVALENT	VITAL CAPACITY IN PER CENT OF TOTAL	TIDAL AIR IN PER CENT OF TOTAL TIDAL AIR	TIDAL AIR IN PER CENT OF VITAL CAPACITY OF OPERATED SIDE	RESERVE AIR IN PER CENT OF TOTAL RESERVE AIR	COMPLEMENTARY AIR IN PER CENT OF TOTAL COMPLEMENTARY AIR
			INTERVAL BETWEEN RE-EXPANSION AND BRONCHO-SPIROGRAPHY	VOLUNTARY EXPANSION	EFFUSION								
42	R	42	55	Yes	+	R: marked fibrosis, retraction. L: mod. advanced	55	2.0	42	60	26	62	30
62	R	3	45	Yes	0	R: practically clear. L: upper thoracoplasty	85	2.1	80	73	20	83	82
69	L	48	36	Yes	0	L: fibrothorax. R: slight apical fibrosis. 3 cm. cavity at clavicle	12	16.4	59	50	28	53	73
70	L	12	5	Yes	+	L: fibrosis upper ¼. R: fibrosis upper ½, cavity 4 cm.	23	5.6	29	37	47		
72	R	12	6	Yes	0	R: mod. fibrosis midzone, slight fibrosis upper ½. L: clear	57	1.6	46	49	13		
74	R	38	9	Yes	Empyema	R: fibrothorax. L: clear	18	8.2	15	45	65	8	6
102	L	24	7	No	Obliterative pleuritis	L: fibrothorax. R: exudative lesion with cavity u. l.	20	4.9	16	34	37	10	14
144	R	9	15	No	Obliterative pleuritis	L: fibrothorax and fibrocaseous lesion in upper half. R: emphysema, few fibrotic lesions between apex and second rib	70	2.3	67	55	20	89	47
153	L	16	3	Yes	0	L: fibrothorax	23	8.8	35	47	31	17	35
	R	8	32	Yes	0	R: min. fibrosis in first i. s.	77	2.9	65	53	19	83	65
235	R	17	0	Yes	0	R: pneumothorax practically disappeared, mediastinum to rt., some fibrosis. L: f. a.	39	5.0	53	62	58	48	39
238	R	35	22	Yes	0	R: mod. advanced	53	4.4	50	54	17	52	47
	L	13	39	Yes	+	L: fibrosis first i. s. (cavity scar) apical fibrosis	47	4.2	50	46	15	48	53

air (in percentage of total tidal air) is much less decreased than the oxygen intake, and in practically all of these lungs the tidal air constitutes a much greater than normal percentage of the vital capacity of the same lung; this may be considered *one* possible rough indication of the restricted ventilatory reserve. In accordance with the observations on other lungs that are functionally damaged it is clear that the respiratory function is almost always more severely impaired than the ventilatory function. The practical conclusion from these observations is that determinations of vital capacity without additional functional tests are totally misleading. Since, as mentioned above, the x-ray appearance is frequently not a reliable indicator of the functional capacity of a lung, it seems to be of great importance to make bronchospirographic determinations before deciding on irreversible collapse procedures, particularly if the contralateral lung has been under pneumothorax treatment.

In our small series of patients it is impossible to correlate the loss of function with such clinical data as length of treatment, development of effusion, and elective re-expansion. Whether or not a re-expanded lung has a considerable loss of function can, at the present state of our knowledge, be decided only by bronchospirographic studies.

Since it was shown in the paragraphs on severely damaged lungs that pleural involvement is the one most frequent factor causing severe functional damage, and since pneumothorax treatment with its potential complications is a frequent cause of severe pleural involvement, this danger to pulmonary function should receive careful consideration when one is deciding upon pneumothorax induction, particularly in patients in whom a selective thoracoplasty can be considered. While our studies, so far, do not permit us to say whether pneumothorax or thoracoplasty produces greater functional limitation, it should be pointed out that in this regard the results of thoracoplasty are more correctly predictable than those of pneumothorax.

COMMENT

The purpose of this report is to show the practical significance of bronchospirographic studies within the field of pulmonary function tests. Bronchographic data, with the exception of the estimation of vital capacity, are not directly comparable with spirometric data, for reasons that were discussed in some detail. Therefore, bronchospirometry is not a substitute but a method complementary to spirometry. Just as in renal function tests it is usually not of interest to determine the function of each kidney separately in diffuse bilateral disease, bronchospirometry has probably little if anything to offer in the functional examination in pulmonary diseases, such as asthma, diffuse (essential) emphysema, and pneumonoconiosis. However, in diseases with

focal disease, either unilateral or asymmetrically bilateral, such as pulmonary and pleural tuberculosis or bronchial carcinoma, it may become important to know the function of each lung separately. The practical necessity of such knowledge arises particularly when irreversible collapse measures (or pneumonectomy) are considered, particularly when the functional capacity of the contralateral side is questionable. Roentgenologic and clinical studies cannot, as was pointed out repeatedly, substitute for functional tests. The functional capacity of a lung may be much poorer, or, at times, much better than its roentgenologic appearance. The former discrepancy is particularly likely to exist in the presence of pleural involvement, the latter in cases of strictly parenchymal lesions. In either case, the results of bronchospirometric studies may decisively influence surgical indications and contraindications.

As a rule, only deficiencies in pulmonary function, have so far received attention in contraindications. Further observations are necessary to determine whether unusually high degrees of compensatory hyperfunction should not be regarded as warning signals as well. High degrees of compensation are indicative of restricted functional reserve, possibly to the extent that further compensation for contralateral loss of function cannot be made.

Through the separate testing of each lung, the degree and type of compensation are more clearly shown than in spirometry; the same applies to the correct evaluation of the limitation of function caused by disease or collapse treatment. How the total function of both lungs may obscure the functional impairment was pointed out in regard to the ventilation equivalent whose real significance is much more clearly shown in bronchospirometric than in spirometric determinations. Essentially the same considerations are indicated in regard to the other ventilatory factors (vital capacity and tidal, reserve, and complementary air) and oxygen intake.

The value of bronchospirometry then becomes evident for surgical indications not only in single patients but for its principal and general application in the more complete understanding of the influance of disease and therapy upon pulmonary function.

In the first portion of this paper, we have presented data for more or less normal lungs. The difficulty and probable impossibility of establishing generally applicable normal standards for bronchography have been discussed. But in spite of this problem these so-called normal values may serve as a guide in comparison with data concerning lungs whose function has been altered by disease and treatment. It is of great practical importance that we were able to show that *mutatis mutandis*, severe pleural involvement, causes not infrequently more profound functional damage than do advanced parenchymal lesions. Since pneumothorax treatment is probably the one most frequent single cause of

extensive pleural disease, this potential danger should receive due consideration, particularly when selective thoracoplasty or strict bed rest are possible alternative procedures. On the other hand, it has been shown that so-called re-expanded pneumothorax lungs may not only function well but may even be capable of considerable compensatory hyperfunction. The functional restriction produced by thoracoplasty, provided it is not complicated by a paralysis of the diaphragm, appears to be fairly well predictable. The functional results of pneumothorax appear to vary so extensively, probably largely dependent on pleural complications, that they cannot be predicted.

TABLE XIII

SIGNIFICANCE OF DIFFERENCES OF MEANS ("P") (OF OXYGEN INTAKE, VENTILATION EQUIVALENT, VITAL CAPACITY, AND TIDAL AIR) BETWEEN NORMAL LUNGS AND (1) SEVERELY DAMAGED LUNGS (PLEURAL INVOLVEMENT), (2) STRICTLY PARENCHYMAL LESIONS, AND (3) THORACOPLASTY LUNGS

	O_2 INTAKE IN PER CENT OF TOTAL	VENTILATION EQUIVALENT	VITAL CAPACITY IN PER CENT OF TOTAL	TIDAL AIR IN PER CENT OF VITAL CAPACITY
Severely damaged lungs (pleural group)	<0.01	<0.01	<0.01	<0.01
Strictly parenchymal lesions	<0.1 >0.05	<0.1 >0.05	<0.3 >0.2	<0.4 >0.3
Thoracoplasty lungs	<0.01	<0.02 >0.01	<0.02 >0.01	<0.05 >0.02

Table XIII is presented to show which figures presented in previous tables in the groups of severely damaged lungs, parenchymal lesions, and thoracoplasty lungs are significantly different from our "normal" group. It is assumed that differences are statistically significant when "P" <0.05.

The severely damaged lungs and the thoracoplasty lungs differ significally in all calculated data from the normal lungs. The parenchymal lesions do not differ significantly from the normal lungs.

We may add to this table the fact that the lungs with parenchymal lesions show so little decrease of function that they differ significantly ($P < 0.01$) from the severely damaged lungs in regard to oxygen intake, ventilation equivalent, vital capacity, and tidal air in per cent of vital capacity.

SUMMARY

1. Bronchospirography is an important method in establishing proper indications for collapse measures; it permits the determination of the extent to which each lung contributes to the total pulmonary function.

2. Bronchospirographic data are presented for apparently normal lungs, severely damaged lungs, lungs with strictly parenchymal lesions, thoracoplasty lungs, and re-expanded lungs.

3. In all lungs that are severely damaged functionally, pleural involvement appears to play an important role.

4. Lungs with strictly parenchymal lesions, even when far advanced, frequently have only slightly decreased pulmonary function.

5. Re-expanded pneumothorax lungs may or may not show extensive functional limitation.

6. Thoracoplasty lungs, if not complicated by diaphragmatic paralysis, participate to a considerable degree in respiration.

7. In lungs damaged by disease or treatment, respiration is usually much more limited than ventilation. It is, therefore, misleading to judge their function by their vital capacity alone.

8. Clinical and roentgenologic studies do not provide a reliable indicator of the functional pulmonary capacity.

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(See discussion on page 287.)

STUDIES OF INDIVIDUAL LUNG FUNCTION

I. An Open-Circuit Procedure With Air and Low-Oxygen Mixtures as the Inspired Gases

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MANY patients who appear to have good respiratory function do not do well following thoracoplasty, while other patients who have manifest pulmonary disability stand the operation well. An adequate explanation of this fact was afforded several years ago by Jacobaeus, who showed that respiration is sometimes supported almost entirely by the lung which needs collapse. Thus, from a functional standpoint, the ultimate outcome of thoracoplasty depends upon the ability of the contralateral lung to carry on gas exchange both at rest and under the stress of physical activity. A comprehensive clinical study of respiratory function, therefore, should include measurement of the response to a stress applied to each lung separately and especially to the contralateral lung. This can be done by giving first one lung and then the other a low-oxygen mixture to breathe while the opposite lung breathes air, in a manner which will be described.

It is possible to predict, at least in part, the response to physical activity by measuring the maximum breathing capacity. The significance of this value has been studied in detail by Cournand, Richards, and Darling[1], who have used it extensively in the estimation of respiratory efficiency. Leiner, Pinner, and Zavod[2] have applied it to the individual lungs. Although maximum breathing capacity is not a measurement of pulmonary circulatory function, it is, nevertheless, one of the most valuable single procedures that it is possible to follow. It is sometimes important, however, to know whether the pulmonary circulation is so distributed that the blood within its capillaries is exposed to a volume of air which is adequate for diffusion both at rest and during physical activity.

Two recent papers by Jacobaeus and Bruce[3] describe a study of the ability of the lungs to substitute for each other when one lung breathes nitrogen and the other lung breathes oxygen, or when the air passages to one lung are blocked while the other lung breathes oxygen. These interesting and instructive papers have so far become available to us only in abstract form and therefore will not be discussed here.

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A straight, rigid bronchoscope of the type used by Jacobaeus and his associates cannot be left in place long enough to make a complete study of gas exchange without causing strain and discomfort. To overcome this difficulty, Gebauer[4] and Zavod[5] introduced flexible rubber catheters containing two channels which effectively separate the air breathed by each lung and which can be left in place as long as local anesthesia lasts. These catheters make possible long periods of observation under reasonably normal conditions.

It has been pointed out elsewhere[6, 7] that the adaptive mechanisms initiated by breathing low-oxygen mixtures are either deficient or ineffective in abnormal lungs and that latent abnormalities may thus be brought to light in the resting subject. In persons with abnormal lungs the arterial oxygen saturation is lower while breathing a low-oxygen mixture than it is in normal subjects, indicating that the absorption of oxygen under stress is limited by the condition of the lungs.

This principle has been applied to the study of individual lung function. A low-oxygen mixture is inspired first by one lung and then the other with the opposite lung breathing air. In this manner one lung at a time is subjected to stress, and the response of both lungs is measured simultaneously.

The pulmonary ventilation and oxygen consumption of each lung are usually measured in small closed-circuit spirometers attached to each channel of the catheter or bronchoscope. With this type of apparatus, each lung rebreathes the gas in a closed system, the valves of which direct the expired gases through a carbon dioxide absorber. The removal of oxygen from the systems by the lungs changes the concentrations of oxygen in the breathing mixtures. If the test is begun with breathing mixtures similar in composition to the atmosphere, the changes in concentration may be too rapid to permit equilibrium between the alveolar air and blood to reach a steady state unless oxygen is added to both systems at the rates at which it is absorbed. This is inconvenient and often technically difficult. If, on the other hand, there is an excess of oxygen in the breathing mixtures at the outset, the changes in oxygen concentration are unimportant but the oxygen-rich atmospheres raise the pressure of oxygen in the alveolar air and favor the absorption of oxygen by inefficiently ventilated lungs. It has not been demonstrated that this materially alters the amount of oxygen absorbed by either lung in relation to the total oxygen absorption, but it is certain that it does not put either lung under an unusual stress.

Since it is hard to keep the composition of the breathing mixtures constant in small closed-circuit spirometers containing low-oxygen mixtures, it has been found convenient to use an open-circuit method in which the inspired air is separated from the expired air without rebreathing. The inspired air can be composed of any desired mixture of gases, and its composition is constant throughout each period of

observation. The expired air of each lung is collected in a suitable container, its volume is measured, and samples are taken for analysis. In addition to the data obtainable by closed-circuit spirometry, carbon dioxide excretion and the respiratory quotient of the expired air of each lung can be determined. These additional data, including the response of each lung to an artificial stress of controllable intensity, would appear to justify the longer period of time necessary for the completion of the test.

Thus, the total gas exchange of both lungs can be measured separately but simultaneously in the resting subject, with both lungs breathing atmospheric air and with a stress in the form of a low-oxygen mixture applied to first one lung and then the other.

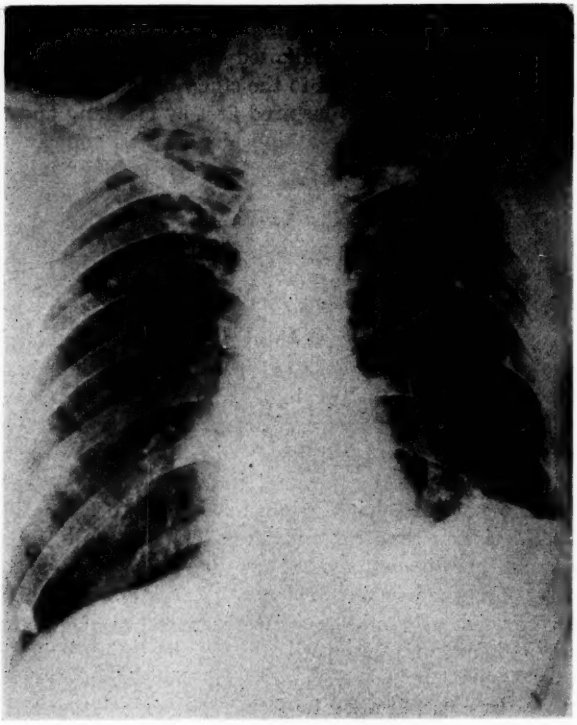


Fig. 1.

CASE 1.—A 26-year-old white man had pneumothorax on the left and a cavity in the apex of the right lung (Fig. 1). It was not possible to collapse the right lung with pneumothorax, and thoracoplasty was therefore considered. The question to be answered was whether the collapsed left lung would be able to support respiration after thoracoplasty on the right.

Fig. 2 shows the more significant results of the test. The black bars represent oxygen absorption, and the white bars, carbon dioxide elimination in cubic centi-

meters per minute per square meter of body surface. In each of three periods of about fifteen minutes, total gas exchange is indicated by the pair of bars designated *T*. This is the gas exchange of the left lung *(L)* added to that of the right *(R)*. In the first period air was breathed by both lungs; in the second period 16 per cent oxygen in nitrogen was breathed by the left, and air, by the right; and in the third period air was breathed by the left, and 16 per cent oxygen, by the right.

The total metabolism was unusually low throughout the test—possibly because a barbiturate had been given in preparation for the anesthetization of the pharynx and upper respiratory tract with cocaine.

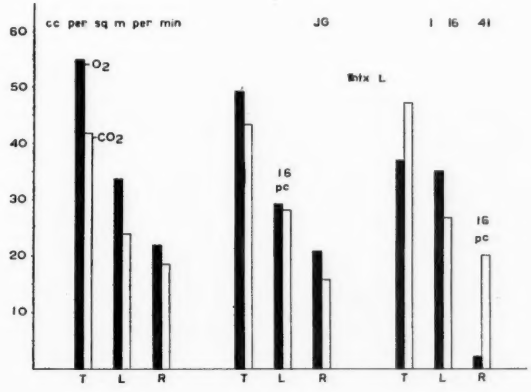


Fig. 2.

The collapsed left lung accounted for most of the gas exchange in all three periods. The function of this lung was not much changed when it inspired 16 per cent oxygen, and there was no compensatory increase in the activity of the right lung during the same period. In the third period the right lung, while breathing the low-oxygen mixture, absorbed a very small amount of oxygen but it excreted more carbon dioxide than it did in any other period. At the same time there was a marked compensatory increase in the function of the collapsed left lung. Because of its ability to maintain gas exchange while breathing a low-oxygen mixture and to compensate for a deficiency in the opposite lung, the collapsed left lung was considered to be much more efficient than the right, and therefore thoracoplasty would cause but little further reduction in the total pulmonary reserve.

In the third period the carbon dioxide excretion of the right lung was greater than its oxygen absorption, indicating that the respiratory quotient of the expired air of this lung was greater than unity while that of the air expired by the left lung was less than unity. This has no metabolic significance whatever, but it does mean that, in relation to the circulation of blood through the pulmonary capillaries, the right lung was being hyperventilated while the left lung was not. The high respiratory quotient of the body as a whole is accounted for by the very high respiratory quotient of the right lung, and it indicates that carbon dioxide was being eliminated from the body more rapidly than it was formed in the tissues. From this it would appear that ventilation was stimulated at this time by a mechanism dependent upon oxygen lack rather than on carbon dioxide excess.

In Table I, the relationship between oxygen absorption and pulmonary ventilation is expressed in the percentages of oxygen removed from the inspired air or gas mixture. Regarded as cubic centimeters of oxygen removed from each 100 c.c. of air inspired, these values have the same significance as ventilation equivalents for oxygen absorption. Abnormally low values accompany overventilation while abnormally high values accompany underventilation. As long as the respiratory quotient of the expired air is a true reflection of the metabolic activity of the tissues, however, the higher the percentage of oxygen removed from the inspired air the more efficient the gas exchange.

TABLE I

LUNG	INSPIRED GAS (%)	O_2 CONSUMED (C.C.)	CO_2 EXCRETION (C.C.)	O_2 REMOVED FROM INSPIRED GAS (%)	CO_2 ADDED TO EXPIRED AIR (%)	R.Q.	MINUTE VOLUME (LITERS)
Right	21	22	18	3.84	3.09	0.80	0.58
Left	21	33	24	4.24	3.09	0.73	0.77
Totals		55	42	4.07	3.11	0.77	1.35
Right	21	21	16	4.25	3.20	0.73	0.50
Left	16	28	27	3.31	3.26	0.96	0.83
Totals		49	43	3.68	3.24	0.86	1.33
Right	16	2	20	0.33	2.95	6.64	0.68
Left	21	35	27	3.79	2.93	0.77	0.92
Totals		37	47	2.31	2.94	1.27	1.60

For description see text.
Gas volumes in units per minute per square meter of body surface.

The relationship between carbon dioxide output and ventilation is shown in the percentages of carbon dioxide added to the expired air. These values may be regarded as ventilation equivalents for carbon dioxide elimination, and they are much more constant than the corresponding equivalents for oxygen absorption. The great difference between the percentages of oxygen removed by the left and right lungs while breathing 16 per cent oxygen without a corresponding change in the percentages of carbon dioxide added to the expired air indicates that a pulmonary capillary bed of sufficient size for carbon dioxide elimination may be inadequate for oxygen absorption. Consequently, an effort to absorb more oxygen by hyperventilation may result in the excretion of an excessive amount of carbon dioxide with a temporary change in the acid-base balance of the blood. It is evident that the disturbance of gas exchange is characterized by an inability to absorb oxygen rather than by an inability to eliminate carbon dioxide.

The respiratory quotient can be obtained by dividing the percentage of carbon dioxide added to the expired air by the percentage of oxygen removed by the inspired air. Since carbon dioxide is eliminated from the body from twenty to thirty times more readily than oxygen is absorbed, overbreathing lowers the percentage of carbon dioxide added to the expired air less than it lowers the percentage of oxygen removed from the inspired air. The numerator of the fraction is therefore reduced less than the denominator, and the quotient rises. The reverse is true in hypoventilation. For there is a difference of only 6 mm. Hg between the carbon dioxide tension of the mixed venous blood and arterial blood, while the difference between the partial pressures of oxygen in the mixed venous blood and arterial blood is about 40 mm. Hg. Thus, when the alveolar air is kept in contact with the pulmonary capillary blood for an unduly long time, oxygen will be absorbed after equilibrium between the carbon dioxide tension of the alveolar air and mixed venous blood has been reached and carbon dioxide excretion has therefore stopped. This situation does not occur,

of course, except in greatly underventilated parts of the lungs, as in pulmonary cysts and emphysematous bullae,[8] but the extreme case may serve to illustrate the effects of underventilation.

The respiratory quotient of the expired air, therefore, gives evidence of either overventilation or underventilation (whichever predominates) in relation to the size of the capillary bed. This information cannot be obtained by measuring the ventilatory volume alone. For it will be seen in Table I that, in the case of the right lung breathing a low-oxygen mixture, the oxygen absorption of that lung was lowest and its ventilation was highest for any period. In spite of the fact that the ventilation of the right lung was less than that of the left, the respiratory quotient of the right lung was 6.64, while that of the left was 0.77. Usually the respiratory quotient varies inversely with the percentage of oxygen removed from the inspired air, but, when there are functionally dead spaces in the lungs, the percentage of oxygen removed will be lowered without changing the respiratory quotient. No amount of ventilation of pulmonary spaces devoid of circulation can change the amount of carbon dioxide excreted or of oxygen absorbed.

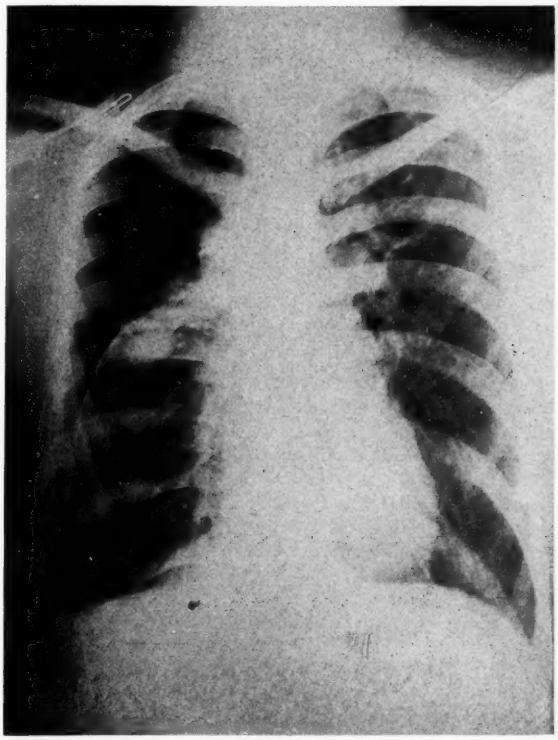


Fig. 3.

CASE 2.—A white male, aged 26 years, had pneumothorax on the right side and a persistent cavity in the upper lobe of the left lung (Fig. 3). Because of the position of this cavity an extensive thoracoplasty would probably be required to bring about healing. Although pulmonary function as a whole had been found to be good, the ability of the collapsed right lung to carry an extra load was investigated prior to collapse of the left.

The results are shown graphically in Fig. 4. It is important in comparing the gas exchange of the two lungs to note that the total metabolism was not constant during the three periods of observation and that this chart is drawn on a smaller scale than Fig. 2.

It is clear from Fig. 4 that the right lung is less efficient than the left, but in the second period, in which the left lung breathed 12 per cent oxygen, there was a definite and marked compensatory increase in the function of the right and this occurred, it will be noted, without an increase in the respiratory quotient of that lung. The reduction of the function of the left lung during this period did not seem to be great, considering that 12 per cent oxygen was being inspired.

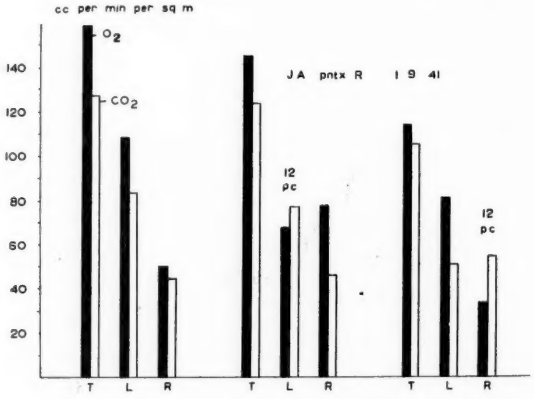


Fig. 4.

In the third period while the right lung breathed 12 per cent oxygen the impairment of function of the right lung is apparent. The rate of oxygen absorption by this lung was maintained by overventilation as indicated by the excessively high respiratory quotient. Nevertheless, during this period the oxygen absorption of the right lung showed about the same relationship to the oxygen absorption of the air-breathing left lung as it did during the first period when both lungs breathed air.

Thus, an extensive thoracoplasty on the left side would permanently deprive the patient of his best lung, but the favorable response of the right lung to a considerable stress would seem to indicate that the left lung could be collapsed if necessary. Of course, the future functional status of any lung collapsed by pneumothorax is doubtful because of the possibility of complicating effusions, pleural symphysis, and the like.

Additional data are included in Table II. The percentages of oxygen removed by both lungs from the total gas inspired $\left(\frac{\text{total oxygen consumed}}{\text{total minute volume}} \times 100\right)$ for each period are not remarkable, but there are great differences between the separate lungs in the relationship between ventilation and oxygen absorption. A high percentage of oxygen removed from the inspired air appears to be a compensatory mechanism during stress because the highest values occur in lungs breathing air while the opposite lung breathes a low-oxygen mixture, but it should be borne in mind that large amounts of oxygen may be removed from the ventilated air without imparting a sufficient amount of oxygen to the venous blood to maintain a normal saturation of the hemoglobin in the arterial blood. This is especially likely to be the case in persons whose ventilatory capacity is reduced and whose functional residual air is large in relation to the breathing capacity.

The high values in Case 2 are accompanied by low respiratory quotients, suggesting longer or more extensive contact between alveolar air and pulmonary capillary blood. However, another factor which might lower the respiratory quotient of a lung removing a high percentage of oxygen from its inspired air is the elimination of an excessive amount of carbon dioxide by the opposite lung at the same time. This would leave less carbon dioxide to be eliminated by the first lung and thus tend to lower its respiratory quotient.

TABLE II

LUNG	INSPIRED GAS (%)	O_2 CONSUMED (C.C.)	CO_2 EXCRETION (C.C.)	O_2 REMOVED FROM INSPIRED GAS (%)	CO_2 ADDED TO EXPIRED AIR (%)	R.Q.	MINUTE VOLUME (LITERS)
Right	21	50	44	4.92	4.34	0.88	1.01
Left	21	109	83	6.03	4.57	0.76	1.81
Totals		159	127	5.64	4.51	0.80	2.82
Right	21	77	46	7.19	4.33	0.60	1.07
Left	12	68	77	3.93	4.43	1.12	1.73
Totals		145	123	5.18	4.39	0.85	2.80
Right	12	33	55	2.53	4.27	1.69	1.30
Left	21	81	51	7.79	4.94	0.63	1.04
Totals		114	106	4.87	4.53	0.94	2.34

For description see text.
Gas volumes in units per minute per square meter of body surface.

CASE 3.—(Fig. 5.) A white female, aged 38 years, had the left lung re-expanded after a two-year period of pneumothorax. The left pleura is thickened; the left diaphragm is paralyzed; and there are several small cavities in the upper third of the left lung. A cavity in the apex of the right lung disappeared during a six-month period of pneumothorax. When the right lung was collapsed, the patient complained of shortness of breath after refills, and, although her dyspnea was not severe, the pneumothorax was abandoned for this reason. Bilateral bronchial catheterization was done to ascertain whether the dyspnea during right pneumothorax was due primarily to an inefficient right lung or to a practically functionless left lung which could not carry a substantial part of the burden of respiration.

This patient had rested on a Bradford frame for a period of a year at the time of the test, and because of muscular weakness she was unable to sit up unassisted. It is difficult to see how an exercise test could have been used at this time. The decision to do a thoracoplasty was made on the basis of the response to low-oxygen mixtures, before a program of treatment which had been beneficial in the past was altered.

Fig. 6 shows that the metabolic activity of the body changed but little during the test and that respiration was supported almost entirely by the right lung. Breathing a low-oxygen mixture had little effect on the function of the right lung. This and the fact that the patient responded normally to breathing both 16 per cent and 12 per cent oxygen with both lungs and without sedatives and local anesthesia on other days would seem to indicate that the right lung is an efficient lung in spite of the history of dyspnea during right pneumothorax. In any event, it would not appear that collapse of the left lung could have much effect on the total respiratory function.

The percentages of oxygen removed from the inspired air and of carbon dioxide added to the expired air, given in Table III, indicate that, in addition to a very low gas exchange, the left lung utilizes but a small part of its ventilation. During the first two periods, in which the left lung breathed air, the percentages of oxygen

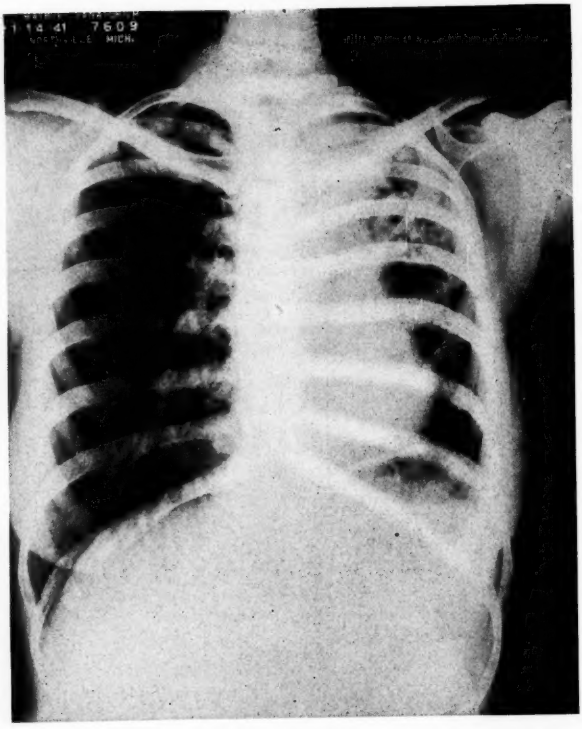
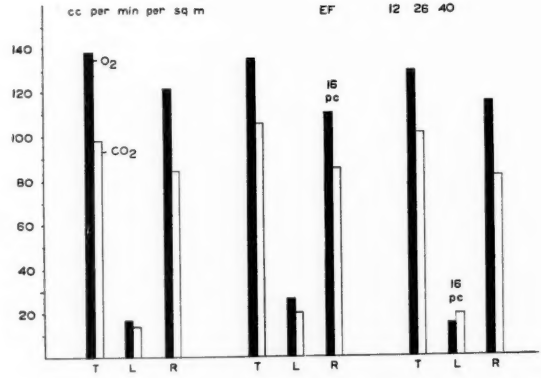


Fig. 5.


cc per min per sq m
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O2
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T L R

Fig. 6.

removed and carbon dioxide added were abnormally low in the presence of a normal respiratory quotient. This indicates that the effective dead space is large and that a large part of the ventilated air did not come into contact with the pulmonary capillary blood.

TABLE III

LUNG	INSPIRED GAS (%)	O_2 CONSUMED (C.C.)	CO_2 EXCRETION (C.C.)	O_2 REMOVED FROM INSPIRED GAS (%)	CO_2 ADDED TO EXPIRED AIR (%)	R.Q.	MINUTE VOLUME (LITERS)
Right	21	121	84	4.48	3.13	0.70	2.69
Left	21	17	14	2.54	2.08	0.82	0.67
Totals		138	98	4.11	2.92	0.71	3.36
Right	16	109	85	4.08	3.20	0.78	2.68
Left	21	25	20	2.88	2.21	0.77	0.82
Totals		134	105	3.83	3.00	0.78	3.50
Right	21	114	82	4.33	3.12	0.72	2.62
Left	16	15	19	1.80	2.23	1.24	0.85
Totals		129	101	3.72	2.91	0.78	3.47

For description see text.
Gas volumes in units per minute per square meter of body surface.

DISCUSSION

An increase in the efficiency of the mechanism by which oxygen is transferred from the alveolar air to the blood has been repeatedly observed while the oxygen tension of the inspired air is reduced. It is thought that increased vascularization of the pulmonary capillary bed occurs when the oxygen tension of the inspired air is reduced, thereby increasing the surface area available for diffusion. New pulmonary capillaries can open when the occasion demands,[9] and a redistribution of blood between the greater and lesser circulations might be the means by which the capillary bed could be enlarged promptly and without increase in the total cardiac output. Hurtado, Kaltreider, and McCann[10] have observed both dilated alveoli and engorged pulmonary capillaries in the lungs of guinea pigs kept for a short period in atmospheric air at reduced pressure. The extent to which these changes favor the absorption of oxygen by inefficient lungs is reduced as a result of disease.

All patients subjected to this procedure have been given a barbiturate —usually 2 grains of phenobarbital the evening before and 2 grains on the morning of the test before the application of cocaine. Some experimental evidence that this alone may, in some persons, produce the picture of hypoventilation in the expired air and a low-oxygen saturation in the arterial blood has already been obtained. While these drugs (and also morphine) may modify the interpretation of this kind of test when considered in terms of general respiratory efficiency under ordinary living conditions, a comparison of the function of one lung with that of the other at the same time and under exactly the same conditions must be valid. The results may then be correlated with further studies of respiratory function made on other days without the influence of drugs.

SUMMARY

The determination of the total gas exchange of the lungs separately but simultaneously by an open-circuit method is a perfectly practical procedure. As compared with the closed-circuit method its disadvantages are that gas analysis is necessary and that more time is required—from five to seven minutes—for each period of observation.

The advantages of the open-circuit method are that it gives more complete data and that the inspired gas, which may be air, or any desired mixture, is constant in composition at all times. This means that any effect of breathing high-oxygen mixtures may be avoided without technical difficulties and that each lung may be subjected to stress separately in the form of breathing a low-oxygen mixture.

It has been demonstrated that the amount of oxygen absorbed by inefficient lungs breathing low-oxygen mixtures is reduced. It might be expected that the amount absorbed by such lungs breathing a high-oxygen mixture would be increased, but this has not been demonstrated.

In every instance studied so far the more efficient lung was that which absorbed the greater part of the total oxygen consumed by the body while both lungs breathed air. From this it might seem that one need proceed no further, but it often happens that the functionally better lung has a cavity in it which must be collapsed if the patient is to be given his best chance of recovery. The ability of the contralateral lung to respond to a stress then becomes a matter of the greatest importance.

It is only occasionally that the decision to do a thoracoplasty depends upon a painstaking physiologic study, but we feel that the future well-being of many borderline patients can be greatly enhanced by knowing in advance how the load of respiration is divided between the two lungs.

The three cases presented were chosen from a series because they illustrate some of the physiologic principles which are important in the selection of cases for thoracoplasty. The methods used permit the quantitative estimation of three practical factors: (1) the performance of each lung at rest without benefit of breathing atmospheres unusually rich in oxygen; (2) the response of each lung to an artificial stress, including the ability of the contralateral lung to compensate for a loss of function on the part of the other lung, and (3) the relation between circulation and ventilation in the individual lungs both at rest and under stress.

Our experience so far seems to indicate quite definitely that this kind of data will not only help to avoid operating on patients who would be made physiologically worse by the procedure but also to extend the benefits of thoracoplasty to some of those thought to be unfit.

Acknowledgment is gratefully made to Leo A. Sheehan, William C. Vogel, and Newton Whitman for technical assistance.

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(See discussion on page 287.)

BRONCHOSPIROGRAPHY

VENTILATION AND OXYGEN ABSORPTION OF NORMAL AND DISEASED LUNGS DURING NITROGEN RESPIRATION IN THE OPPOSITE LUNG

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IT HAS been shown[1, 2] that, when a lung is collapsed, its respiratory functions are diminished and those of the contralateral lung are increased. Occasionally the ability of the pulmonary tissue to increase its function is so poor that anoxemia develops and, if the collapse is irreversible, death may ensue.

The ability of a lung to take up oxygen depends chiefly on four factors: (1) the volume of blood perfusing it, (2) the oxygen tension of the venous blood coming to it, (3) the oxygen tension of the alveolar gas (which is in turn dependent upon the alveolar ventilation), and (4) the diffusion characteristics of the alveolar membranes. An increase in (1) and (3) and a decrease in (2) will augment oxygen absorption. Changes of an opposite sense or the development of an abnormal degree of resistance to oxygen diffusion at the alveolar membrane will result in a decrease of oxygen absorption. In collapse therapy an increase of alveolar ventilation and volume of perfusing blood is the chief means of augmenting oxygen absorption by the contralateral lung. Failure of the proper response of one or all of these factors results in respiratory crippling or death.

The aim of all pulmonary function tests is to supply evidence which will allow one to determine beforehand the ability of the functioning tissue to take over the work of the collapsed tissue. Such tests are of two general types: (1) those measuring the maximum functioning capacity (reserve) of both lungs together and (2) simultaneous measurement of the part contributed by each lung to the resting pulmonary function (bronchospirography). If the subject's respiratory reserve is good and the diseased organ contributes only a small per cent to the total function, the patient can afford the loss of the diseased lung with but little further pulmonary incapacity. Conversely, a patient with a low reserve, a large portion of which is accomplished by the lung to be collapsed, might find himself in severe difficulty as the result of an irreversible procedure. The limits, as shown by measurements of func-

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tion, at which serious or fatal respiratory incapacity will result from collapse therapy, have not been satisfactorily determined.

This paper deals principally with an attempt to determine the capacity of the contralateral lung to take up increased amounts of oxygen when oxygen absorption by the side to be collapsed is blocked by respiring pure nitrogen. We do not believe that such a procedure simulates collapse therapy except in that it will cause an increase in the volume of oxygen which passes the membranes of the contralateral lung and hence might be used as a method of testing its ability to perform this task. The blood perfusing the side respiring pure nitrogen not only picks up no oxygen but actually gives it off, and, when this blood of low-oxygen tension mixes with the normally oxygenated blood from the opposite lung, arterial blood of a low-oxygen tension is produced. Since there is no reduction in tissue oxygen utilization, the oxygen tension of the venous blood coming to the lung is lowered. The degree of lowering of the arterial and venous blood oxygen tensions depends mainly upon the volume and oxygen tension of the blood flowing from the side blocked by nitrogen (nitrogen respiring lung). If the reduction in arterial oxygen tension is sufficient, chemoreceptors in the aorta and carotid bodies will be stimulated, resulting in an increase in ventilation and an increase in cardiac output. In the presence of a normal alveolar membrane, these factors provide the mechanism for an increase in oxygen absorption from the lung respiring room air. The differences between the response to oxygen block and to pulmonary collapse are chiefly that in the latter there is an actual shunt of blood from the collapsed lung to the contralateral one and also there is no excretion of oxygen from the collapsed pulmonary tissue such as occurs in the lung breathing nitrogen.

What might be the response in persons with pulmonary disease? Nitrogen block of a lung accomplishing only a small percentage of the total oxygen consumption should produce but little drop in arterial oxygen tension and, therefore, no increase in ventilation and but little increase in oxygen absorption from the opposite side. If the blocked lung accomplishes a sufficient portion of the total oxygen absorption, there should be a marked fall in arterial oxygen tension and an increase in ventilation and oxygen pick-up from the opposite side. Theoretically one might expect the response of the side breathing room air to be in direct proportion to the percentage of oxygen utilization accomplished by the lung to be blocked with nitrogen. Abnormalities present in the thoracic bellows or parenchyma of the contralateral lung, which might militate against its ventilating power, the efficiency of its ventilation, an increase in the minute volume of blood perfusing it, or the ease with which oxygen diffuses across the alveolar membrane, should limit the response on the part of the lung breathing room air below that to be expected. The degree of response might also be

affected by the narcotics used in preparing the patients for study since these drugs decrease to a varying degree the sensitivity of those centers controlling ventilation and cardiac output.

TECHNIQUE

In these experiments it is essential that the gases ventilating each lung be kept entirely separate. For this purpose we have used the catheter devised by Gebauer[3] with one modification. The balloons on his catheter (as supplied by the American Cystoscope Company) do not distend equally because of their thick irregular walls. We have replaced them with ones having very thin walls (thickness of condom rubber) made by dipping glass molds into liquid latex. These balloons distend evenly and equally at pressures of 2 to 4 cm. of mercury. We have further modified Gebauer's technique for passing the catheter and making measurements of lung function as will be apparent in the following.

The patient comes to the laboratory in a fasting state and, after a thirty-minute rest period, the minute ventilation, oxygen consumption, carbon dioxide output, maximum minute ventilation, and vital capacity are measured. The subject is then given 1.5 grains of nembutal. Thirty minutes later this dose is repeated, followed in ten minutes by 0.25 grain of morphine sulfate and 0.01 grain of atropine. This dosage is varied according to the history of the patient's sensitivity to narcotics. We desire to use the least degree of narcosis which will permit the measurements to be made without discomfort to the patient. One hour after the first nembutal is given the pharynx and tracheobronchial tree are anesthetized with 5 per cent larocaine and the catheter is passed under observation with a laryngeal mirror, the subject being in a sitting position. When the catheter is well engaged in the trachea, the patient lies on the fluoroscopy table and the tip of the catheter is advanced into the left main bronchus while being observed. The balloons are inflated and the catheter connected through Bailey mercury valves to the two spirometers containing room air. A one- to two-minute tracing is made for the purpose of checking on the position of the catheter. Following this short tracing the spirometers are disconnected from the valves and the subject breathes room air for four to five minutes, after which a six-minute collection of expired gas is made in Douglas bags, both lungs breathing room air. The side to be collapsed is then caused to breathe pure nitrogen from an anesthesia bag, while the other lung continues to respire room air. A two-minute period is allowed to elapse for the development of a low-oxygen tension in the organ being ventilated with pure nitrogen, following which a six-minute collection in Douglas bags is made from each lung. The side to be collapsed continues to inspire pure nitrogen throughout this collection. The gases are measured in a 100 liter spirometer and analyzed in duplicate for oxygen and carbon dioxide on the Haldane apparatus. The oxygen consumption and carbon dioxide output are calculated from this data according to the method introduced by Tissot and described in detail by Boothby and Sandiford.[4]

In nine of the thirty-four patients studied, the arterial oxygen saturation was measured before catheterization, with the catheter in place and both lungs breathing room air and also with one side breathing pure nitrogen. The blood samples were collected in mercury sealed containers and stored on ice. They were analyzed within three hours on the Van Slyke apparatus.

DISCUSSION OF TECHNIQUE

The Tissot technique for measuring oxygen absorption and carbon dioxide output was adopted for two reasons. In experiments (Table I) performed on normal

dogs in which one lung respired pure nitrogen and the other lung respired pure oxygen, we found that the arterial oxygen tension dropped only slightly. Apparently the extra 3 c.c. of oxygen (oxygenation of remaining hemoglobin plus oxygen in physical solution at higher tension) which can be taken up by the blood perfusing the lung respiring pure oxygen decreases the deficit in arterial oxygen tension caused by the mixture of blood from the lung breathing nitrogen. These observations were later confirmed by the report of Bruce and Jacobaeus.[5] These investigators found that in human beings, when one lung breathed pure oxygen and the other breathed pure nitrogen, the arterial oxygen saturation dropped to only 90 per cent or 86 per cent. The fact that the lung breathed nitrogen in a closed circuit in their experiments and in ours the lung breathing nitrogen exhaled to the outside means that the alveolar air in the side breathing nitrogen in their experiments had a higher oxygen tension than in ours. The dog experiments (Table I) show this to be a minor factor however. We believe that our observations cast a doubt upon the determination of differential pulmonary blood flow by methods in which one lung breathes pure oxygen.[5, 6]

TABLE I

LUNGS	SUBSTANCE BREATHED	ARTERIAL O_2 CONTENT (VOL. %)	ARTERIAL O_2 CAPACITY (VOL. %)	SATURATION (%)
Both (with bronchoscope in place)	Room air	17.8	19.0	94
Right	Room air	13.7	19.0	72
Left	100% N			
Right	100% O_2	17.3	19.0	91
Left	100% N			
Both	100% O_2	20.8	19.0	109

A rigid double bronchoscope designed to avoid blocking the left upper lobe bronchus was used in this study.

Since we wanted to lower the arterial oxygen tension as much as possible, we were forced to abandon any procedure using a high-oxygen mixture in the lung to be tested. Room air, although of the proper oxygen content, cannot be used in a spirometer with a closed circuit for any length of time because of the progressive decrease in oxygen tension. The Tissot technique, though laborious, fulfilled our requirements. Duplicate runs with an interval of five minutes between gas collections show the experimental error with the Tissot technique to be in the neighborhood of 1 per cent, as performed in our laboratory.

A second reason for adopting the Tissot technique is that measuring the oxygen consumption from the slant of a tracing obtained by rebreathing in a spirometer is frequently made difficult and inaccurate by irregular breathing. Such irregular breathing is quite common in subjects with a bronchial catheter in place. A comparison in forty-one cases of the oxygen consumption as calculated by the gas analysis method and from the two-minute tracings obtained from the spirometer at the beginning of an experiment is seen in Table II. The wide variation (5 per cent or more in one lung) between the results obtained by the Tissot technique and the spirometer tracing in twenty of the forty-one cases strongly suggests that the spirometer technique is not altogether trustworthy. In those cases in which breathing was very regular, there was adequate correlation, but in none of the irregular breathers was this true. It is to be emphasized, in fairness to the spirometer technique, that the correlation might be better if tracings of more than two minutes' duration were used. However, in a number of cases where such

tracings were available for comparison, the correlation still left much to be desired. The Tissot technique possesses the added advantage of allowing one to study the carbon dioxide output.

It is worth noting (Table III) that, when a subject breathes room air through the catheter, there is a slight drop in arterial oxygen saturation, suggesting that the airway is not sufficiently large to provide perfect ventilation. In Case 22 it is possible that the left upper lobe was blocked, as is suggested by the drop in arterial oxygen saturation from 91 per cent to 85 per cent. When a gas of high-oxygen tension, such as is used in the spirometer technique, is respired through the catheters, this drop in arterial oxygen saturation does not occur.

TABLE II

NO. OF CASES	DIFFERENCE IN PER CENT OF O_2 ABSORBED BY ONE LUNG AS DETERMINED BY TISSOT AND SPIROMETER TECHNIQUES
3	No difference
10	1% to 2%
8	3% to 4%
6	5% to 6%
11	7% to 15%
3	Too irregular to calculate from spirometer tracing
41 total compared	

TABLE III

CASE NO.	O_2 CONSUMPTION BLOCKED (%)	INCREASE IN VENTILATION (%)	INCREASE OF O_2 ABSORBED FROM ROOM AIR LUNG (C.C.)	ARTERIAL O_2 CAPACITY (VOL. %)	ARTERIAL HEMOGLOBIN SATURATION (%)		
					WITHOUT CATHETER	CATHETER IN PLACE	ONE LUNG, ROOM AIR; OTHER, 100% N
47	5.0	9.0	5.0	19.4	92.0	90.0	88.0
40	25.0	9.0	33.0	20.0	94.0	90.0	76.0
7	32.0	27.0	49.0	18.1	93.0	90.0	67.0
44	39.0	30.0	120.0	18.5		94.0	73.0
42	42.0	19.0	142.0	19.7		91.0	77.0
30B	42.0	78.0	302.0	21.2	96.0	90.0	72.0
26	47.0	53.0	122.0	19.7	95.0	93.0	69.0
30A	53.0	60.0	182.0	21.7	93.0	90.0	67.0
50	58.0	97.0	195.0	20.9	86.0	85.0	62.0
22	64.0	49.0	234.0	22.9	91.0	85.0	69.0

RESULTS AND DISCUSSION

The thirty-four cases reported in this study had all been accepted on a clinical basis for collapse therapy prior to our observations. Although a few were considered to be of the less desirable type of risk, the degree of damage in the contralateral lung was not extensive in any instance. The absence of severe damage in the contralateral lung in the cases being presented in this report must be taken into account when evaluating our results.

A scatter graph showing the relationship between the percentage of total oxygen consumption accomplished by the lung blocked with

nitrogen and its ventilation response is shown in Fig. 1. The ventilation, in terms of per cent of increase above that observed when both lungs respired room air, is plotted on the ordinate. The percentage of total oxygen consumption (both lungs breathing room air) which was blocked by nitrogen is plotted on the abscissa. The condition of the contralateral lung being tested (breathing room air) is designated by one of three symbols. A triangle represents a case having no x-ray

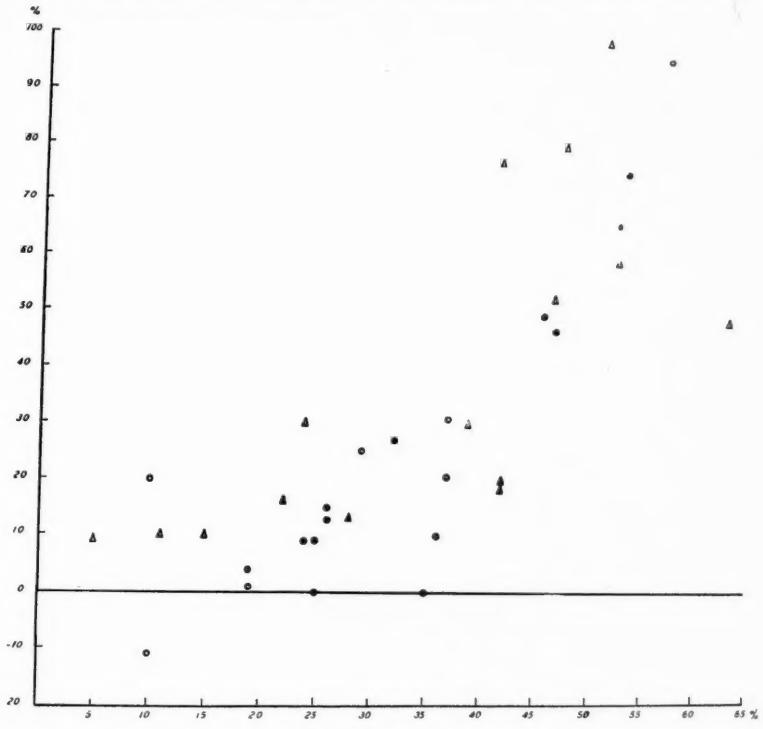

Fig. 1.

evidence of disease in this lung. An open circle represents a lung with a single small cavity, a small infiltration or nonprogressive fibrotic disease of less than one-fourth of it. A solid circle represents a lung which at the time of the study showed evidence of pulmonary changes involving one-fourth or more of the parenchyma or demonstrable changes in the thoracic bellows apparatus. The presence of abnormalities did not necessarily indicate active disease at the time of the study. It is apparent (Fig. 1) that no correlation exists between the condition of the lung tested and the ventilation response, nor is there a close correlation between the per cent of oxygen block and the ventilation response. When more than 40 per cent is blocked, there is a greater

increase in ventilation but there is still a wide variation in the amount of increase at all levels. This is more strikingly shown in Table III. Cases 7 and 30A show the same per cent of arterial oxygen saturation when one lung respires nitrogen (indicating similar arterial oxygen tensions), but widely different ventilation responses (27 per cent and 60 per cent, respectively). The most likely explanation of the variability of the ventilation response seems to be that under the conditions of these experiments there is a difference in sensitivity of those centers responsible for bringing about ventilation changes.

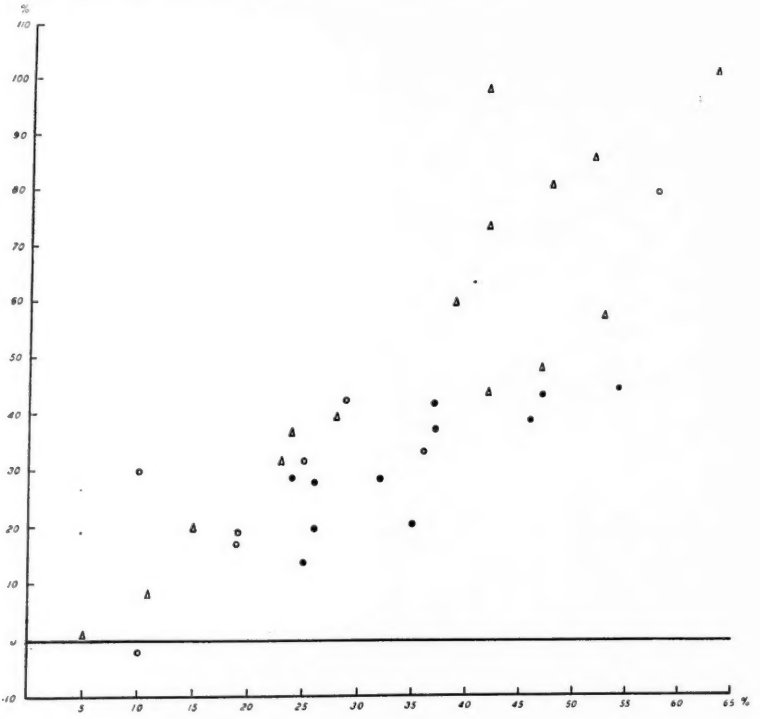

Fig. 2.

In Fig. 2 (using the same symbols) the increase in oxygen taken up by the lung breathing room air is plotted on the ordinate as the percentage which this increased volume represents of the total oxygen consumption for both lungs when breathing room air. The abscissa is the same as in Fig. 1. The grouping of the damaged lungs along the lower border of the scatter suggests some failure on their part to attain the proper degree of response. The presence of three healthy lungs in close proximity to the diseased ones detracts from the value of this observation. The correlation between the percentage of oxygen blocked

by nitrogen and the increase in oxygen picked up by the side breathing room air is much better than that for ventilation response, but there is still a wide scatter at any one level.

The variable reaction to blocking a certain percentage of the total oxygen consumption (viz., the four normal lungs along ordinate *40* on the abscissa of Fig. 2) suggests that this response involves rather complex mechanisms. Unfortunately we have been unable to repeat our observations on the same person under identical conditions on different days, and hence we do not know what variations might normally occur. A comparison of Case 30A with 30B (Table IV) brings out some of the factors that help one to understand why variations among normal persons might be expected to occur. Case 30B is the same as Case 30A studied after paralysis and before paralysis of the right hemidiaphragm. The left lung appears to be normal. Before the paralysis (30A), blocking 53 per cent of his total oxygen consumption (right lung) resulted in a drop of arterial blood saturation to 67 per cent, an increase in ventilation of 60 per cent, and an increase of 182 c.c. per minute in oxygen absorption from the left lung. After paralysis (30B), blocking 42 per cent of his total oxygen consumption (right lung) resulted in a drop in arterial blood saturation to only 72 per cent, an increase in ventilation of 78 per cent and an increase of 302 c.c. per minute in oxygen absorption from the left lung. Theoretically it would seem that the responses should have been reversed, the largest increase in ventilation and oxygen absorption of the left lung being associated with the largest percentage of oxygen block.

TABLE IV

CASE NO.	O_2 CONSUMPTION WHEN BREATHING ROOM AIR	O_2 BLOCKED BY N (%)	VENTILATION INCREASE (%)	O_2 INCREASE (c.c.)
30B	290	42	78	302
12	255	42	20	116
30A	310	53	60	182
15	260	54	76	121

This apparently paradoxical response occurring in the same person might be explained as follows. The fact that he ventilated more in response to the smaller percentage of oxygen block must mean that on the day he was studied after the paralysis the nerve centers controlling the ventilating response were either naturally more sensitive, or were less depressed by the narcotics used, as compared with the day he was studied before paralysis of his diaphragm. The much larger increase in the volume of oxygen picked up by the left lung (breathing room air) in response to the lesser percentage of total oxygen consumption blocked during the study after the paralysis involves two factors. The increase in ventilation not only raises the alveolar oxygen tension in the side breathing room air (left) but also materially aids in blowing off oxygen from the side breathing pure nitrogen with a resultant lowering of

arterial blood oxygen tension. Both of these factors bring about an increase in oxygen absorption from the lung breathing room air and consequently will tend to give the greatest increase in oxygen picked up by the side breathing room air when there is the largest increase in ventilation. A striking example of this mechanism is shown by comparing Cases 30B and 12 in Table IV. The percentage of oxygen block is equal in both cases, but the increase in oxygen absorption in Case 30B is nearly triple that in Case 12. It is significant that the ventilation increase in Case 30B is also triple that in Case 12. The other factor contributing to the explanation of the variations in response in Case 30A and 30B is as follows. If the arterial blood is normally saturated with oxygen when both lungs breathe room air, the percentage of total oxygen consumption accomplished by one side represents roughly the percentage of total cardiac output circulating through that lung. In this instance 47 per cent of the blood coming to the lungs was perfusing the left one (30A) before the paralysis, whereas 58 per cent was perfusing this same side after the paralysis (30B). Under such conditions, in spite of what must have been a higher level of oxygen tension in the blood returning to the left lung after the paralysis (since less oxygen was blocked and the arterial blood saturation is actually higher), more oxygen was picked up by this side because of the greater minute volume of blood perfusing it. Two opposing factors are in operation. The larger the percentage of oxygen absorbed by one lung, the larger its portion of circulating blood, and hence when it is blocked with nitrogen, the greater will be the fall in blood oxygen saturation. By the same token, however, the opposite side, having less blood perfusing it, will have less capacity for increasing the amount of oxygen that it can absorb.

A comparison of Case 15 with Case 30A in Table IV suggests that a lung which is the site of an appreciable amount of disease is hampered in its ability to increase its oxygen absorption. In Case 30A the side respiring room air is normal, whereas in Case 15 the lung respiring room air was not only diseased but moderately collapsed by a pneumothorax. Although the same oxygen percentage was blocked in both, Case 15, in spite of an advantage in ventilation increase, was unable to increase his oxygen absorption as much as Case 30A, either on a percentage basis or in absolute figures.

SUMMARY AND CONCLUSIONS

When one lung breathes room air and the other breathes pure nitrogen, there is an increase in the amount of oxygen picked up by the side respiring room air and in most instances there is also an increase in ventilation. The increase in ventilation exhibited by the lung respiring room air shows very little direct relation to the percentage of total oxygen consumption blocked by nitrogen or to the degree of

lowering of the arterial blood saturation. We believe this to be due to the natural variations in sensitivity of the nerve centers controlling ventilation and to the depressant action of narcotics upon these same centers.

We wish to emphasize the fact that the mechanisms brought into play by these experiments are not identical with those which occur when one lung is collapsed by a surgical procedure. We have attempted to increase the amount of oxygen picked up by the lung opposite the one to be collapsed in an effort to detect some disability on its part to accomplish this task. Although the diseased lungs appear to show less ability to increase their oxygen absorption than do normal ones, the difference is not as striking as we had expected. It is possible that a more clear-cut incapacity might be revealed by more extensively damaged lungs.

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DISCUSSION ON "BRONCHOSPIROGRAPHY" BY DR. MAX PINNER, DR. GEORGE LEINER, AND DR. WILLIAM A. ZAVOD;* "STUDIES OF INDIVIDUAL LUNG FUNCTION" BY DR. WALTER K. WHITEHEAD, DR. E. J. O'BRIEN, AND DR. WILLIAM M. TUTTLE;† AND "BRONCHOSPIROGRAPHY" BY DR. GEORGE W. WRIGHT AND DR. WARRINER WOODRUFF

DR. SAMUEL A. THOMPSON, New York, N. Y.—For years we have been using bronchial catheterization as a diagnostic procedure as well as a therapeutic procedure. The adaptation of bronchial catheterization to bronchospirography seems to be entirely satisfactory and may perhaps simplify the procedure somewhat, because one of the assets of bronchial catheterization is the ease with which the bronchi may be catheterized. As a matter of fact, it may be done as an office procedure.

I have two or three slides which I should like to show.

This shows the set-up for bronchial catheterization. It is simply an apparatus for the local anesthesia, with a special laryngeal tip, a laryngeal mirror, and the bronchial catheters. In addition, there is an inflation tube or an occlusion cuff which slips over the end of the catheter, making it possible to block completely the bronchus when this is desired.

*See page 241 for article by Pinner, Leiner, and Zavod.
†See page 266, for article by Whitehead, O'Brien and Tuttle.

This picture shows a bronchial catheter in the right upper lobe, a single catheter in one lobe.

This shows a bronchial catheter in the right upper lobe and, at the same time, another bronchial catheter in the left lower lobe. This demonstrates how more than one lobe may be catheterized simultaneously.

This shows the catheter in the right upper lobe and another in the left lower lobe, at the same time.

Here we have a catheter in the right upper lobe, another in the right middle at the same time.

This is an oblique view of the previous slide.

I should like to suggest to the men who are doing this type of work that the technique of bronchospirography be extended to the point where it is possible to ascertain the function of each individual lobe, rather than the function of one lung against another.

DR. HERBERT C. MAIER, New York, N. Y.—I should like to illustrate a few of the things that may be learned by bronchospirography which has been discussed by the essayists.

(Slide) In this case we have a patient with a left pneumothorax of one year's duration, in which there is a marked thickening of the pleura without empyema, but with a pulmonary cavity remaining at the apex. Obviously it is an ineffective pneumothorax. Before this lung was collapsed, there was no disease in the lower portion of the left lung. The bronchospirographic tracings indicate what poor function there is on the left side, illustrating one of the points made by Dr. Pinner, that, due to pleural complications, one may have serious impairment of the function of a lung.

(Slide) These are the data taken from that case. I merely wish to point out that the ventilation on the left side is very small as compared with that on the right, and the oxygen intake of the left lung is so small that it could not be accurately measured. The arterial oxygen saturation was 93 per cent while both lungs were breathing room air. When we had the left lung breathe nitrogen by the method mentioned by Dr. Wright, we noted little change. In other words, there is very little function on this left side.

(Slide) In contrast to the previous case, in which we would say that the disturbed function on the left was rather obvious from the roentgenogram, we have here a patient who had had a pneumothorax on the right side of five years' duration, with some pleural fluid at one time. She now has multiple cavities at the left apex which require thoracoplasty.

The question was, "How much function exists on each of these two sides?" Here we note on the spirographic tracing that we have good function in this reexpanded right pneumothorax and moderate ventilation persisting on the left side.

(Slide) The data from this same case show a moderate ventilation on the left and a further diminished oxygen intake. In this particular case, when the lung we were going to collapse was breathing nitrogen, there was a moderate reduction in the arterial oxygen saturation, indicating that, although we could proceed with thoracoplasty on the left, there was still a moderate function of this left lung, and therefore as much of the base of that lung should be conserved as possible.

(Slide) The one film is taken in inspiration and the other film in expiration, indicating obstructive emphysema on the right. From the ordinary film taken in inspiration only, we could hardly have judged that the right lung was functioning very little and that the left lung was doing most of the ventilation.

The data in that case show the marked impairment of ventilation on the right side and the still greater reduction in oxygen intake of the right lung. Here, too, when the right lung breathes pure nitrogen, there is only a moderate change in the arterial oxygen saturation.

(Slide) This is a case in which a left upper lobe lobectomy had been done two years previously. We note that there was fairly satisfactory ventilation on that left side, namely, of the left lower lobe.

(Slide) The further information on the case shows that the remaining left lower lobe was performing approximately one-third of the total ventilation and about one-third of the oxygen intake and, as a matter of fact, is actually more efficient than the right lung.

In this case, because the left lower lobe is doing a considerable amount of work, we note a marked reduction in the saturation of the arterial blood when the left lower lobe breathes nitrogen.

I show these cases as further illustrating some of the points that may be studied by the methods that have been discussed.

DR. EDWARD J. O'BRIEN, Detroit, Mich.—I believe we should all be very much pleased to have heard these splendid papers this afternoon. I believe they are tremendously important to all of us as thoracic surgeons.

As Dr. Whitehead said, "It is in only a small group of patients that it would be necessary to make these observations." When it is necessary, however, it is very necessary. In our clinic we feel that we have salvaged a few patients (some that we thought could not withstand the operative procedure), and we have been prevented from operating upon others who probably would have died had thoracoplasty been performed.

When this study was first started, we applied the usual criteria for operative indication and then checked our results with preoperative lung function tests. We have arrived at a point now where, in borderline cases, we would not think of subjecting a patient to thoracoplasty without obtaining the figures on the respiratory function of each lung separately.

Our old idea of lung function, as a whole, has to be revised because (as has been shown), in some instances, the apparently damaged and functionless lung is carrying most of the burden. The supposedly good or contralateral lung often has markedly diminished function. In these patients, thoracoplasty on the supposedly crippled lung might be fatal.

DR. MAX PINNER.—I should like to say a few words about the other two papers of this afternoon—first about Dr. Whitehead's presentation. I should like to ask Dr. Whitehead whether he has any definite information, or any data, to show that having a lung breathe a low percentage of oxygen is equivalent to putting that lung under physiologic stress similar to that which occurs when the patient exercises. The actual physiologic equivalent of breathing a low-oxygen mixture would be to send the patient to the mountains. I wonder if he has data to show that breathing low-oxygen mixtures is a reliable test which runs somewhat parallel to exercise tests.

I am very much interested that practically all of the speakers entirely agreed with me that it is impossible by clinical and roentgenological evidence to foretell accurately, in many cases, the actual functional ability of the lung. The scattering of the response that Dr. Wright obtained when one lung was blocked with nitrogen might be thought of in two different ways. It is possible that the irreg-

ular response is due to undetected disease in the contralateral lung, such as obliterative pleuritis, so easily not detected by x-ray examination. On the other hand (and this is an important question in regard to the method as such), is not the response of the normal lung determined to a large extent not only by the functional ability of that lung but by the amount of disease in the lung that is blocked by nitrogen? In other words, if the lung that is blocked has very little function, then, of course, the response of the other lung cannot be very marked.

The criticism of bronchospirometry against gas analysis is entirely correct. In bronchospirometry, the accuracy of the determination of oxygen consumption lags behind the accuracy in gas analysis. We have the impression that if we choose for actual use only those records which show a fairly good curve, the accuracy is good enough for clinical use, probably not for research use such as has been reported by Dr. Wright and Dr. Whitehead.

I am glad that Dr. O'Brien agrees, too, that this method is important for collapse therapy. I should like to go one step further. I believe it is not only important from the point of view of deciding whether a patient can safely be operated upon or not. As I tried to indicate in my paper before, I believe we have here a method which will eventually help considerably in deepening our knowledge about certain physiologic functions of respiration, and I am quite certain that since we have done this work we have not only made occasionally a more intelligent selection of patients for collapse therapy, but have actually learned more about the physiology of respiration.

DR. WALTER WHITEHEAD, Northville, Mich.—We have used low-oxygen mixtures as a means of producing stress, primarily in order to avoid exercise, because the response to exercise is dependent on physical fitness and it is not confined to the respiratory system alone.

We have not compared the response to exercise to that produced by breathing low-oxygen mixtures, although I believe we should. It is possible that exercise may increase the surface area for diffusion, the gas-blood interface, more effectively than low-oxygen mixtures, but there is little doubt that the mechanism by which oxygen is transferred from the alveolar air to the blood is more efficient in normal than in abnormal lungs.

To the best of my knowledge, the open-circuit method has as wide an application as the closed-circuit method, and we have been very much pleased with the accuracy and additional data it affords.

DR. GEORGE W. WRIGHT, Saranac Lake, N. Y.—Our experience with bronchospirography is in agreement with Dr. Pinner in that the thing which is most damaging to pulmonary function is not the parenchymal lesion but rather the disease process which impairs normal function of the bellows mechanism. Limitation of motion on the part of the thoracic cage and the diaphragm, usually resulting from pleural disease, accounts for most of the respiratory damage we see. Of course extensive parenchymal disease in itself reduces pulmonary function, but it is nearly always accompanied by severe impairment of the respiratory bellows mechanism. In cases where there is parenchymal disease with no damage to the bellows apparatus we find little or no respiratory damage demonstrable by bronchospirometry. This does not mean that a degree of respiratory incapacity fails to exist since the bronchospirometric procedure alone measures only the part played by each lung in a comparative resting condition and does not measure the reserve respiratory capacity.

We agree with other investigators that the roentgenogram alone is often misleading in giving one an estimate of the amount of pulmonary disability present. Together with Dr. Sampson we have routinely made a careful fluoroscopic study

of our subjects, paying particular attention to the motion of the diaphragm and thoracic cage. From these observations we have been able to make more accurate estimates of the relative role in respiratory function played by each lung than was possible from the roentgenogram alone. The case just shown by Dr. Maier bears this out. Unless I am mistaken, the films of this case showed the mediastinum to shift to the right on inspiration and to the left on expiration in the classical manner of obstructive emphysema of the right lung. In view of this it is not surprising that the right lung, though appearing to be relatively free of disease in a single roentgenogram, actually contributes but little to the total pulmonary function.

TREATMENT OF ENDOBRONCHIAL TUBERCULOSIS

A Review of 100 Cases

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INTRODUCTION

ONE hundred consecutive cases of endobronchial disease complicating pulmonary tuberculosis have been studied. All lesions were in the trachea or major bronchi and were easily seen through the bronchoscope. They were of such severity that their presence was not only indisputable, but one could predict with certainty that irreversible damage had resulted. All doubtful cases of mucosal reddening and edema or questionable submucosal lesions were excluded.

CLASSIFICATION

The cases were divided into two large groups based on the stage of the bronchial disease. They were either acute or chronic. The acute cases were further divided into three types—(1) hyperplastic, (2) ulcerative, and (3) ulcerohyperplastic. The chronic cases were those which had progressed to the smooth fibrostenotic end stage and were subdivided into (1) mild, (2) moderate, and (3) marked stenosis. The analogous bronchus in the contralateral lung was used as a basis for comparison. Accurate classification of the acute cases was difficult because often only the proximal border of the lesion was seen since the dangers from extensive exploration are out of all proportion to the information gained.

PHYSIOPATHOLOGY

Endobronchial tuberculosis is essentially a problem of mechanical obstruction in a patient with pulmonary tuberculosis, and obstructive emphysema, bronchiectasis, and atelectasis are therefore frequent complications of this disease.

In the acute phase of endobronchial tuberculosis improper drainage of the diseased area occurs because bronchial peristalsis is reduced, ciliary action is lost, and the lumen is narrowed by granulomatous tissue or caseous exudate.

In the chronic phase of the disease there are two types of obstruction. In both the mucosa is smooth and intact. In the first type there is localized cartilaginous weakening with probable destruction which results

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in exaggerated mobility of the bronchial wall so that on inspiration the lumen may be normal in diameter, but on expiration its walls collapse into a narrow slit. Obstructive emphysema is the result. The second type of chronic obstruction is the inelastic stricture of the bronchus known as the fibrostenotic lesion in which the diameter of the lumen is only slightly influenced by respiratory movements.

Since both acute and chronic endobronchial tuberculosis cause obstruction, it is fair to assume that the physiologic morbidity from such obstruction is directly proportional to (1) the location of the involved bronchus, (2) the degree of the stenosis, and (3) the duration of the obstruction. When a prolonged, severe obstruction occurs in the stem bronchus, almost complete loss of respiratory function may be the result.

Atelectasis and bronchiectasis resulting from retained secretions contribute to the decreased respiratory function, but air is also imprisoned and the emphysema which results may so dominate the picture that the amount of dyspnea is noted to be out of all proportion to the parenchymal lesion. A variable amount of obstructive emphysema is present in all of these cases and becomes of increasing significance the greater the obstruction. The role of obstructive emphysema is not often emphasized.

STATISTICAL DATA

The number and distribution of the patients in regard to the kind of therapy may be seen in Table I. The patients were listed according to the predominating form of therapy since, obviously, many of the pneumothorax or thoracoplasty patients were receiving bed rest also. The patients limited to bed rest therapy form the largest group, but many of these were pneumothorax failures, had refused thoracoplasty, or were not eligible for collapse therapy because of age, amount of pulmonary tuberculosis, or extrapulmonary contraindications.

TABLE I

ONE HUNDRED CASES OF ENDOBRONCHIAL TUBERCULOSIS

	BED REST	PNEUMOTHORAX	THORACOPLASTY	CAVITY DRAINAGE	PNEUMONECTOMY
Apparently arrested	4	3	18	0	0
Improved	6	4	4	1	1
Unchanged	21	3	2	1	0
Worse	10	5	1	2	0
Dead	5	7	0	2	0
Total	46	22	25	6	1

The conspicuous figure in Table II is the mortality rate of 33⅓ per cent in twenty-two cases treated by pneumothorax. Progression of the pulmonary disease and tuberculous empyema (eleven cases) were the main complications contributing to this figure.

In Table III eighteen of the patients are negative to concentrate examinations. Fourteen of the eighteen are also negative to repeated cultural examinations, but four have shown an occasional positive culture.

TABLE II

PNEUMOTHORAX RESULTS IN ENDOBRONCHIAL TUBERCULOSIS

(22 CASES)

	NO. OF CASES	
Apparently arrested	3	33⅓%
Improved	4	
Unchanged	3	33⅓% (Approximately)
Worse	5	
Dead	7	33⅓%
Total	22	

TABLE III

THORACOPLASTY RESULTS IN ENDOBRONCHIAL TUBERCULOSIS

(25 CASES)

	NO.	PER CENT	
Apparently arrested	18	72%	88%
Improved	4	16%	
Unchanged	2	8%	12%
Worse	1	4%	
Dead	0		0%
Total	25		

Two of the four in the improved group have recently had their operations and appear to be doing well. One patient still has endobronchial disease, and another is still positive after a revision operation. The condition of this patient is excellent, and lobectomy seems indicated.

Two have remained unchanged, and one is not only worse, but will most certainly die from his disease and surgical complications.

Not only are the majority of the patients negative to laboratory examinations, but they have proved to be physiologically capable of returning to society.

DISCUSSION

The management of cases complicated by severe endobronchial disease in the large bronchi is based on one's understanding of the physiopathology of bronchial obstruction. The familiar statement that the best treatment of endobronchial tuberculosis is through control of the parenchymal lesion is only of significance if one realizes the added difficulty of such an accomplishment. Endobronchial tuberculosis tends to run a self-limited course,[2, 10] and therefore the "healed" fibrostenotic end stage is often seen in the presence of an uncontrolled parenchymal lesion. But a stenosis sufficient to embarrass the cleansing mechanism of the lung is consistently followed by irreversible complications, namely, emphysema, atelectasis, and bronchiectasis.

Therapy directed toward the pulmonary lesion must be one step ahead of these complicating sequelae. The first task in management is early recognition of the disease.[9] A negative bronchoscopy in the presence of convincing evidence of endobronchial tuberculosis simply localizes the disease beyond the bronchoscopic field. When a severe acute lesion is discovered in a major bronchus, the experienced bronchoscopist may predict with considerable accuracy the stenotic end result and therapy must be established with this end in view.

Reduction in the size of the pulmonary bed should be considered when the amount of obstruction or the amount of anticipated obstruction is determined. Collapse of the lung is desirable because the pulmonary lesion is more likely to heal and such a lung is less vulnerable to secondary suppuration.

Occasionally endobronchial tuberculosis is not diagnosed until after the institution of collapse therapy. If it is thought that the amount of collapse is sufficient to control the parenchymal disease but the sputum remains positive, bronchoscopic examination and local therapy may be indicated. The stagnation of secretions in pneumothorax is thought by some[3, 8, 12] to be of pathogenic importance. Others[1, 13, 15] have noted the development of endobronchial tuberculosis after thoracoplasty.

TREATMENT

Local Therapy.—Treatment should be directed toward both the bronchial and parenchymal lesions. We have used topical applications of silver nitrate with improvement in many cases, but we do not wish to describe this improvement as a cure in the true sense of the word. Better bronchial drainage and a reduction in the vascularity of the granulation tissue is the rule, and often that part of the bronchial disease visible through the bronchoscope will disappear.

Pneumothorax.—In general we are opposed to pneumothorax as a therapeutic procedure in severe disease of a large bronchus. Several authors[4, 11, 16] have reported good results in that the patients showed symptomatic improvement and conversion of sputum, but the ultimate outcome of these cases in regard to re-expansion and the status of the pleural cavity is not yet recorded. Our own results were poor. An ineffective pneumothorax[14] with suspending adhesions will, of course, give neither of the desired results, i.e., prevention of suppuration or control of the parenchymal lesion, and encourages all of the complications, namely, tuberculous or mixed empyema, suppuration, and spread of the disease.

Nontuberculous suppurative chest diseases have contributed to our understanding of this problem. A lung abscess with poor bronchial drainage rarely heals without surgical aid because air and secretions are retained, allowing the cavity to enlarge, and the inflammatory process ap-

proaches the lung periphery. If it were not for the symphysis of the pleurae, the peripleuritis, and the defensive support of the chest wall, pleural complications would be more frequent. Yet the lung abscess with poor bronchial drainage is not greatly unlike tuberculosis with cavitation and endobronchial disease, but pneumothorax is no longer recommended in the therapeutic program of lung abscess.

Pathologists seem to agree that in all cases of cavitation some degree of endobronchial ulceration may be demonstrated in the draining bronchus. The bronchus in such a case is a small one. Pneumothorax therapy in our opinion should be reserved for these, or perhaps for very mild cases of endobronchial disease in the major bronchi.

If bronchial disease is discovered or develops after pneumothorax has been in effect, one must be careful in re-expanding the lung since to re-expand is to increase the size of the pulmonary bed. If a change of therapy is indicated, it is probably better to do the thoracoplasty over the pneumothorax rather than open up new areas for suppuration through re-expansion.

Drainage.—External drainage of the pulmonary cavity is occasionally necessary. This may be done by the Monaldi technique or by rib resection. We have had one case with bilateral pulmonary disease that also developed severe bronchial disease which was discovered in the acute phase. When local therapy failed, the treatment consisted of Monaldi drainage. This procedure was dramatic in controlling the bronchial lesion. When stenosis is marked and a fulminating secondary infection with abscess formation is superimposed, we have had to resort to rib resection and drainage.

Lobectomy and Pneumonectomy.—Collapse by either pneumothorax or thoracoplasty is occasionally ineffective in converting the sputum. Lobectomy or pneumonectomy may be indicated in selected cases.[5, 6] Such a procedure is still in the experimental stage, but certain fundamentals seem apparent. First, one must rigidly exclude all active tuberculosis except in the lobe or lung to be excised. Second, the bronchus to be severed must be "healed" and free of active disease to prevent a tuberculous empyema or a bronchial fistula. Third, subsequent to pneumonectomy, overexpansion of the remaining lung is undesirable since reactivation of old disease in the remaining lung may occur. If thoracoplasty has not been done previously, it should be done soon after the pneumonectomy.

Thoracoplasty.—Thoracoplasty is the most effective solution to this problem.[7] It will control the pulmonary lesion in the majority of cases. It reduces the size of the pulmonary bed to a minimum, and it eliminates any pleural complications. Success from radical surgery is dependent upon one's effectiveness in overcoming the stagnation of bronchial secretions during the active surgical interval.

The preoperative preparation should include all efforts to improve the patient's general health and muscle tone as well as local treatments of silver nitrate to aid bronchial drainage and cut down vascular granulation tissue. The operation should be done in small stages and preferably under local anesthesia to preserve an effective cough mechanism. The postoperative routine is directed at the prevention of atelectasis by frequent coughing, intratracheal catheter suction, or bronchoscopic aspiration when necessary. A vigilant nursing personnel is indispensable to a successful outcome.

CONCLUSIONS

1. The problem of endobronchial tuberculosis involving the large bronchi is essentially one of mechanical obstruction and its complications (emphysema, bronchiectasis, atelectasis, etc.) in a patient with pulmonary tuberculosis.

2. Early bronchoscopic recognition of the bronchial disease and reduction in the size of the pulmonary bed before chronic stenosis occurs are of fundamental importance in the therapeutic program.

3. Pneumothorax is a dangerous form of therapy when the bronchial disease is severe and large bronchi are involved.

4. Local application of silver nitrate will often aid therapy directed at the pulmonary lesion.

5. Severe endobronchial tuberculosis produces permanent damage to the bronchus and is accompanied by irreversible sequelae. A permanent form of therapy is therefore indicated. The results from thoracoplasty have been good. External drainage, lobectomy, and pneumonectomy may be used in selected cases.

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(See discussion on page 321.)

TUBERCULOUS STENOSIS OF THE MAJOR BRONCHI

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ALMOST a decade has passed since Eloesser,[1] in an excellent discussion of bronchial stenosis, described it as the stepchild of clinical medicine. It is certainly true that our knowledge of stenosing bronchial lesions and their effect on poststenotic disease was then meagre. It is still true that knowledge and experience in these matters is not abundant. Several years were to pass until the early work on tuberculous tracheobronchitis[2-4] began to appear and bronchoscopic examinations became matters of routine. It was only slowly and after numerous disappointments in treatment that we came to understand better the management of stenotic bronchial lesions and the parenchymal changes which the diminished bronchial lumen engendered.

Tuberculous stenosis of a major airway presents a twofold problem. In the ulcerative state its treatment and proper management are often difficult. At the same time, the altered situation created in the parenchyma peripheral to the narrowed bronchial lumen must be considered and dealt with properly. In the past few years we have become better accustomed to dealing with the endobronchial lesions, but it is only very recently that we have begun to realize that special considerations are needed in the appropriate management of the parenchymal disease which many of these patients have.

The bronchus in a tuberculous patient may be narrowed intrinsically by the scarring produced when an ulcerative process in its wall heals, or from without, by the pressure of large tuberculous mediastinal glands. The latter situation is seen most commonly in children and leads, even in those cases in which the caseous node ruptures and drains endobronchially, to no permanent bronchial injury. As the glands recede, the bronchial lumen becomes normal.

This study deals only with bronchial obstruction which has been caused by intrinsic stenosis as a result of ulcerative tuberculous tracheobronchitis. The data here tabulated are the result of a statistical study of ninety-two patients who had tuberculous stenosis of the major

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bronchi. The diagnosis of this type of tuberculosis was made in all instances previous to May, 1940. Thus, all patients have been observed for at least one year.

The stenoses observed have been classified as either ulcerous or fibrous in type and shall be referred to in that manner. Of the ninety-two patients observed, sixty-nine had ulcerostenosis of a major bronchus and twenty-three had fibrous stenosis.

The fibrous stenosis received no treatment unless the poststenotic accumulation of secretions made it necessary to dilate the stenosis. Treatment of the ulcerostenotic type was carried out in the manner previously described as being routine in this clinic, namely, the topical application of 30 per cent silver nitrate at intervals of from two to three weeks. This was done to hasten healing of the tuberculous ulcerations. When, if healing had taken place, it was necessary to dilate the stenosis, this was then carried out in a manner similar to that already described as being used in the patients whose stenoses were of the fibrous type.

Sixty-nine patients with tuberculous ulcerostenosis of the bronchus were treated topically with silver nitrate (30 per cent). Healing of the ulcerated lesion occurred in forty-eight patients, or 70 per cent of the group. Eight patients (11.9 per cent) showed no or only slight improvement after long periods of treatment, and in thirteen (16.4 per cent) the bronchial lesion became definitely worse. The latter state was manifested by extension of the ulcerative process, either to other areas of the primarily involved bronchus, to the trachea, or to the contralateral bronchus.

TABLE I

STATUS OF 69 PATIENTS WITH ULCEROSTENOSIS OF THE BRONCHUS

STATUS	NO. OF PATIENTS	PERCENTAGE
Arrested	18	26.0
Apparently arrested	4	5.7
Quiescent	4	5.7
Unstable	20	28.9
Dead	23	33.3

Tuberculous ulcerostenosis of a bronchus is a serious disorder. The morbidity is great, and the fatality rate is high. Of the original sixty-nine patients, twenty-three (33.3 per cent) are now dead. As may be seen by referring to Table I, the number of cases unarrested far exceeds those classified as arrested.

In Table II, in which the causes of death in the ulcerostenotic group are tabulated, it will be seen that fourteen out of a total of twenty-three deaths (58.3 per cent) were due to poststenotic suppuration and extension of the bronchial disease. In four other instances in which the bronchial lesions healed, the bronchial lumen was reduced to a point where adequate elimination of bronchial and parenchymal secretions was

impossible and poststenotic suppuration resulted in extensive destruction of the pulmonary parenchyma and death. Two patients with ulcerostenotic lesions which healed showed great reduction in the size of the bronchial lumen. This, coupled with extensive parenchymal damage in the contralateral lung, so limited the pulmonary ventilation that death ensued. The remaining four deaths were related only partially to the bronchial lesion.

TABLE II

CAUSES OF DEATH IN 23 PATIENTS WITH ULCEROSTENOSIS OF MAJOR BRONCHUS

CAUSE OF DEATH	NO. OF PATIENTS	PERCENTAGE
Poststenotic suppuration and progressive bronchial disease	14	58.3
Inadequate pulmonary reserve	2	8.6
Poststenotic suppuration	3	13.0
Thyroid crisis	1	5.0
Postoperative thorocoplasty	1	5.0
Tuberculous meningitis	1	5.0
Tuberculous empyema	1	5.0

TABLE III

STATUS OF 23 PATIENTS WITH FIBROUS STENOSIS

STATUS	NO. OF PATIENTS	PERCENTAGE
Arrested	5	21.7
Apparently arrested	1	5.3
Quiescent	3	13.0
Unstable	7	30.0
Dead	7	30.0

The group of patients who had fibrostenosis when first observed did little better than the ulcerostenotic group. Death was due, in the majority, to the same factors which caused death in the ulcerostenotic group, with the exception of poststenotic suppuration. Although the presented series is relatively small and of questionable statistical value, the patient with healed stenosis appears to face great uncertainty.

Poststenotic suppuration which has accounted for more deaths than any other single cause in this series is a serious complication. It is devastating in its destruction of lung tissue. Apparently its effectiveness is due to the symbiotic action of the tubercle bacillus and a facultative anaerobic *Streptococcus viridans*. Anaerobic cultures were made of the sputum in fifteen instances, and in each this organism was found. It is not uncommon under such circumstances to observe a whole lung to become airless and then rapidly excavate with the formation of multiple large and small abscesses. The sputum increases to several hundred grams a day and, when excavation is complete, the amount does not abate. Great difficulty is experienced in eliminating such sputum through the narrowed bronchial lumen. The temperature is almost constantly elevated. The patient wastes rapidly, and death, in the majority, comes in from one to two months after the onset of symptoms.

Frequent dilatation of the bronchus with aspiration gives moderate relief. Blood transfusion has been used extensively but is only of general supportive value. Vaccines of the aspirated organism have been of no value. Chemotherapy has likewise failed. Of a total of twenty-seven patients who had definite evidence of poststenotic suppuration, twelve recovered. The fatality rate was 55.5 per cent.

The treatment of the parenchymal lesion in cases complicated by tuberculous bronchial stenosis has been studied. It would appear that proper handling of the pulmonary lesion is quite as important as adequate care of the bronchus. Collapse therapy was instituted in a certain number of these patients. Pneumothorax was used in forty-seven patients on the side of the stenosis. Thoracoplasty was done in thirty-five patients on the affected side. Phrenic nerve surgery was used as a primary procedure in only ten cases. Five of these developed atelectasis. The number of patients is not large enough to have statistical significance, but the impression gained is that, where stenosis is high grade and the amount of secretion is large, a paralyzed diaphragm is not desired.

TABLE IV

STATUS OF PNEUMOTHORAX IN 47 PATIENTS

STATUS	NO. OF PATIENTS	PERCENTAGE
Tuberculous empyema	15	31.9
Unexpanded lung	22	46.7
Pneumothorax given up	14	29.7
Pneumothorax adequate	12	25.5
Bronchial fistula	2	4.2

Pneumothorax was not a wholly satisfactory collapse procedure (Table IV). From a total of forty-seven patients, who had pneumothorax, in only twelve instances may the procedure be considered as adequate to control the parenchymal disease. In twenty-two patients the pneumothorax was abandoned because of atelectasis, empyema, or inability to control the pulmonary lesions by its use. Fifteen patients developed tuberculous empyema, and in two of these a bronchial fistula with a mixed infection empyema was present. Twenty-two patients had lungs which would not re-expand. Thirteen of a total of thirty-four patients who had no treatment other than pneumothorax are dead. The causes of these deaths are listed in Table V.

When Table V is reviewed, it will be noted that only one patient is listed as having died of tuberculous empyema. Seven of the patients listed as having died of poststenotic suppuration also had empyema which contributed to their death, but it appeared that the primary cause of death was the former rather than the latter. The fate of the pneumothorax appears to depend largely upon the degree of stenosis.

Thus, the twelve patients who did well with pneumothorax had an average bronchial stenosis of 53 per cent. Those who developed atelectasis and unexpanded lungs had an average stenosis of 70 per cent.

TABLE V

CAUSES OF DEATH IN 13 PATIENTS WHO WERE TREATED WITH PNEUMOTHORAX

STATUS	NO. OF PATIENTS	PERCENTAGE
Poststenotic suppuration (uncontrolled bronchial disease)	8	61.5
Poststenotic suppuration (ulcer healed)	1	7.7
Inadequate pulmonary reserve	2	15.4
Cardiac decompensation	1	7.7
Empyema	1	7.7

The group who had thoracoplasty did better (Table VI) than did the patients subjected to other forms of collapse therapy. Of the total group of thirty-four, five are dead; one death was caused by poststenotic accumulation of secretions and inadequate cardiorespiratory reserve ten days following the second stage operation. One patient in whom the parenchymal lesion had been controlled for eight months following thoracoplasty died of tuberculous meningitis. One patient had a first stage thoracoplasty in the presence of a rather severe poststenotic suppuration; it was impossible to complete the thoracoplasty, and three months following the first stage operation she died from the extreme toxicity of the suppurative process. Two patients in this group died two and three years, respectively, following thoracoplasty because of inadequate cardiorespiratory reserve. The remaining patient was never able to tolerate more than one stage of thoracoplasty. She died approximately 2.5 years after the operative procedure of chronic tuberculous empyema (for which the thoracoplasty was done originally) from amyloid disease and an inadequate cardiorespiratory reserve.

TABLE VI

STATUS OF 34 PATIENTS WITH BRONCHIAL STENOSIS TREATED BY THORACOPLASTY

STATUS	NO. OF PATIENTS	PERCENTAGE
Arrested	12	35.3
Apparently arrested	5	14.4
Quiescent	3	8.8
Unstable	9	26.4
Dead	5	14.4

Cavities were present in all patients on whom thoracoplasty was done. The cavities were closed in twenty-eight patients. The immediate fatality rate is 2.8 per cent, and the total fatality rate for the group is 14.1 per cent. The sputum was converted in twenty-five instances. Twelve of the group may now be called arrested; five are apparently arrested; and three are quiescent. Nine patients are still unstable. Of the unstable group, two have recently reopened cavities beneath the

thoracoplasty and will need revisions. Four have apparently controlled bronchial and parenchymal disease, but this occurred too recently to warrant their removal from the unstable group. Two have controlled parenchymal lesions but have persistent bronchial ulceration. In the remaining patient, we have never been able to complete the operative procedure.

DISCUSSION

Tuberculous stenosis, whether the primary lesion or complicating parenchymal disease, is a matter of serious import. As will be noted by referring to Table VII, 32.6 per cent of the entire group are dead. A remaining one-third of the group is unstable, and at least half of these may be considered as hopeless. It seems relatively safe to say that approximately one-third of any group of patients with ulcerostenosis have a chance to become arrested in time. Treatment with silver nitrate (30 per cent) has been used, we believe, with success. Seventy per cent of the ulcerative lesions healed, resulting in a healed stenosis. In those cases which healed with stenosis, the stenotic lesion was in all instances present when the lesion was first seen. In our experience, silver nitrate tends to increase the size of the bronchial lumen by the removal of granulation tissue. There can be little doubt that many ulcerative or ulcerostenotic lesions of the bronchus heal without treatment. However, it seems probable that they heal by stenosis only when the original lesion is of the type which encircles the bronchus. Those bronchial lesions which are confined to a single wall of a bronchus heal with only slight signs of scarring. The ulcerostenotic lesion is extensive when first seen and, as a general rule, covers a fairly large area of the bronchus.

TABLE VII

STATUS OF 92 PATIENTS WITH BRONCHIAL STENOSIS

STATUS	NO. OF PATIENTS	PERCENTAGE
Arrested	23	25.0
Apparently arrested	5	6.6
Quiescent	7	7.3
Unstable	27	28.5
Dead	30	32.6

Myerson[5] has stated recently that none of his reported 160 patients have died as a result of the immediate effects of the bronchial lesion. One cannot be exactly sure what is meant by this statement, but it has been taken at its face value. Certainly this is not our experience or that of many others who see and treat these lesions. Nor has it been either their or our experience, as it has been Myerson's, that all tuberculous lesions of the bronchus heal spontaneously. A sizable group, even with treatment, remain unhealed.

Dilatation of bronchial stenoses is only a palliative measure. Such lesions remain open following dilatation for varying lengths of time, in

many instances, no longer than from a few days to several weeks. The main virtue of dilatation rests in its ability to relieve the immediate obstruction. By its repeated use, coupled with aspiration of secretions distal to the stenosis, the status of the distal bronchus often improves, until the secretions become less in amount and thin enough to pass the stenosis without difficulty.

When the lumen of the bronchus is decreased in size and secretions are blocked, the natural therapeutic endeavor is directed at diminishing such secretions. Secretions which are bronchial in origin cannot easily be stopped. Secretions which have their origin in the lung parenchyma may be stopped by control of the parenchymal disease.

Pneumothorax has been and still is used apparently far too often in the case of bronchial stenosis. The results of our series in which pneumothorax was used are, in the main, poor. The incidence of empyema was high, as was the incidence of lungs totally collapsed and unexpanded. That the stenotic process is in no small way responsible for this is demonstrated by the fact that, in the twelve cases of pneumothorax where this procedure was adequate, the average degree of stenosis was 53 per cent as compared with an average of 70 per cent in those cases which did poorly. From this, one might conclude that the integrity of the bronchial lumen is essential to the success of pneumothorax. The high incidence of lungs which will not re-expand was obviously the result of the bronchial obstruction in many instances.

The high instance of empyema may find explanation in the fact that a large percentage of patients with ulcerative lesions of the bronchus also have varying amounts of exudative disease in the lung. In the presence of a stenosis the sudden collapse of the lung which follows the induction of pneumothorax will give rise to empyema in much the same manner as a pneumothorax which has been instituted over exudative disease and handled injudiciously. Furthermore, many of these pneumothorax spaces are large because of extensive lung collapse. Large spaces with extensively collapsed lungs seem more prone to be the site of empyema than do smaller spaces.

Thoracoplasty obviates by its very nature many of the above objections to pneumothorax. If the collapse of the lung because of stenosis is to be permanent, then the collapsing agent other than the stenosis should be permanent. Thoracoplasty does not violate the pleural space, and the danger of empyema is obviated.

The results in the thoracoplasty group are much better than the pneumothorax group. This may be due in part to the fact that in several instances the bronchial lesion was under better control in the group than when the pneumothorax was instituted. However, whenever the pneumothorax which was already showing signs of giving trouble was re-expanded and thoracoplasty was done, the results have been satisfactory. Such results from the standpoint of therapeutic

collapse of the lung parenchyma would seem to argue for the early establishment of thoracoplasty in such cases.

From a purely theoretical standpoint, removal of the lung distal to the stenosis would seem to be the ideal approach to the problem. Actually, experience with this type of procedure has not been productive of encouraging results. Dolley and Jones[6] last year reported four cases in which this had been done. Two are dead. The remaining two are not well and in the opinion of the authors would never be well. In the discussion which followed their presentation, nineteen other patients subjected to pneumonectomy were presented. Of this group, only three patients are now considered well. Eight of the group were dead, a fatality rate of 40.2 per cent. These figures do not compare favorably with those presented in this study. We have experienced with careful handling but little difficulty in the execution of thoracoplasty in the patient with bronchial stenosis. Dilatation of the bronchus a few days before the operative procedure is to be carried out is generally sufficient to maintain an adequate bronchial lumen during the immediate postoperative stage. Only in a few instances has bronchoscopy been necessary in the first few postoperative days to remove secretions.

When all factors are considered, the end result of these two groups appears to indicate that, when the degree of bronchial stenosis exceeds 50 per cent, thoracoplasty is the procedure of choice to control the parenchymal lesion. Such disasters of therapy as have been related here can be avoided only by greater ability in the diagnosis of early bronchial ulcerostenosis and by greater care in the choice of the therapeutic collapse procedure. In all instances in cases of this type the bronchus must dictate the treatment which the lung will receive. Knowing the dangers which the undiagnosed case of ulcerostenosis may bring forth, one is tempted to advise a routine bronchoscopic examination before pneumothorax is induced in the same manner as we now use such examinations previous to thoracoplasty and other major collapse procedures. Such a suggestion, if carried out, would add greatly to the duties of the thoracic surgeon or endoscopist. However, it is equally as certain that a number of patients in this series might be alive and well today had the status of the bronchial lumen been better understood.

CONCLUSIONS

Ninety-two patients with ulcero- or healed bronchial stenosis have been studied.

Thirty-two per cent of this group are dead. Thirty-three per cent are still classified as unstable.

Sixty-nine patients had ulcerostenosis when first seen. The ulcerative lesion was treated with 30 per cent silver nitrate. Seventy per cent of the group healed while 30 per cent did not change or become worse.

Pneumothorax as a method of controlling the parenchymal processes has been compared with thoracoplasty.

Pneumothorax was adequate in only twelve of the forty-seven instances in which it was used. The incidence of empyema and unexpanded lungs is high (31.9 per cent and 46.7 per cent, respectively).

Thoracoplasty has yielded results superior to pneumothorax.

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(See discussion on page 321.)

EFFECT OF THORACOPLASTY UPON PULMONARY TUBERCULOSIS COMPLICATED BY STENOTIC TUBERCULOUS BRONCHITIS

Incidental Remarks on Pneumonectomy and Lobectomy

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UNTIL ulcerative and stenotic tuberculous bronchitis was widely recognized several years ago as an important clinical entity, the treatment of pulmonary tuberculosis seemed relatively simple. The unexpected and unexplained poor results of treatment in certain cases were accepted as troublesome and were attributed to bad luck. The rapidly increasing use of bronchoscopy in patients presenting signs and symptoms that experience has shown are suspicious of a complicating tuberculous bronchial lesion has led to a sound explanation of the poor results that occur in certain types of tuberculous bronchitis and to a gradually developing knowledge of the form of treatment best suited to different types of bronchial lesion complicating parenchymal tuberculosis.

Sufficient experience has not accumulated to permit the giving of final answers to the many problems presented by tuberculous bronchitis complicating pulmonary tuberculosis. The opinions expressed in this article are based upon the observations of the thoracic surgery staff of the University of Michigan Hospital during its ten-year period of great interest in the subject. These opinions are tentative and probably will be modified on the basis of increasing experience and recommendations from other clinics, as they have been in the past. At present we are certain only of the fact that the presence of tuberculous bronchitis confuses the prognosis and greatly complicates the problem of treatment of pulmonary tuberculosis. The recognition of tuberculous bronchitis as a relatively frequent (approximately 11 per cent) complication of parenchymal pulmonary tuberculosis is probably the most important advance that has been made in the field of tuberculosis during the last decade.

We are opposed to the use of any form of collapse therapy for pulmonary tuberculosis in which there are suspicious signs or symptoms of tuberculous bronchitis until bronchoscopy has been performed in order to

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determine the type, position, and extent of the bronchial lesions, if present. In this article we shall discuss chiefly the relationship between tuberculous bronchitis and thoracoplasty, pneumonectomy, and lobectomy.

Many patients with tuberculous bronchial lesions have such extensive parenchymal lesions that no collapse therapy operation, except thoracoplasty, could reasonably be expected to be effective. The emphasis in the few articles[3, 8, 13] which have been written on the treatment of this combination of bronchial and severe parenchymal lesions has not been upon collapse therapy but upon total pneumonectomy and lobectomy, particularly when the tuberculous bronchial lesions have produced a relatively high degree of bronchial stenosis with secondary atelectasis. Eloesser in 1940 expressed his strong disapproval of thoracoplasty in these cases.

The factors that should determine the choice between thoracoplasty and pneumonectomy (or lobectomy) in the group of patients under discussion are many. Those factors that should, in our opinion, contraindicate pneumonectomy or lobectomy have not been heeded sufficiently in many of the reported cases, and the capacity of thoracoplasty to bring about healing of the lesions has been, we believe, insufficiently recognized.

Facts of great practical importance should be noted. Nineteen cases of pneumonectomy for tuberculosis were reported in the discussion of Dolley and Jones' article.[3] Eight of these patients (40.2 per cent) are dead; two are expected to die; six are improved; and only three are considered well. Thirty-one lobectomies were reported in the discussion of the same article. Eight of these patients (20.5 per cent) are dead; the result in one is "uncertain"; six patients are improved; and sixteen are well (in ten of these sixteen patients, tubercle bacilli were not found in the sputum before the lobectomy operations). In our group of thirty-eight cases of thoracoplasty in the presence of tuberculous bronchitis the results are far better. Only three patients (7.9 per cent) are dead; ten (26.3 per cent) living patients have positive sputum (three being positive only by culture); and twenty-five (65.8 per cent) have apparently completely closed cavities and negative sputum. The patients were followed for from several months to five years after operation. While the thoracoplasty and pneumonectomy-lobectomy groups are not strictly comparable with regard to the severity of the bronchial and parenchymal lesions and of the patients' illnesses, they are sufficiently comparable to make the difference in the results, especially the death rates, of great practical importance.

The solution of the problem is, however, unfortunately not so simple as the mere choosing of the operation having the lowest mortality and producing the highest percentage of healed tuberculosis. A certain small group of patients have a combination of bronchial and parenchymal lesions that cannot be safely or effectively treated by thoraco-

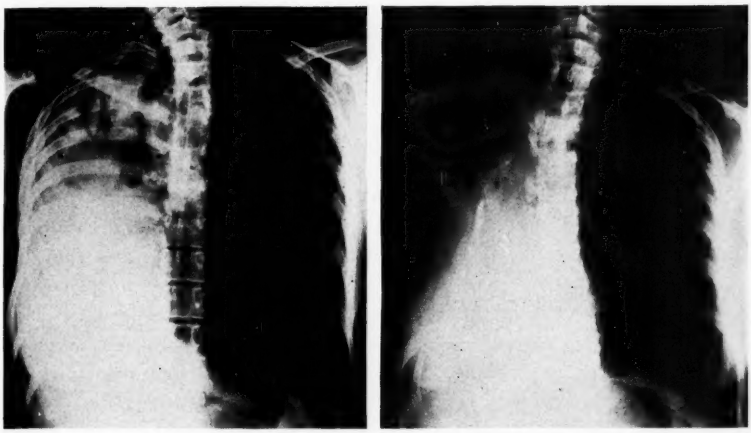
plasty, or a good thoracoplasty may already have been performed without producing a satisfactory result. For such patients pneumonectomy or lobectomy may be the only procedure capable of restoring health. The safest and most effective treatment, however, for the great majority of patients having advanced bronchial and parenchymal tuberculosis is, in our experience, thoracoplasty. We shall now discuss the indications for pneumonectomy, lobectomy, and thoracoplasty, and the special factors that pertain to thoracoplasty in such cases.

Total pneumonectomy is indicated when a tight and bronchoscopically undilatable stricture has produced prolonged toxic symptoms from the retention of purulent secretions in an atelectatic, fibrotic lung that may or may not contain open tuberculous cavities and bronchiectases. Curiously, the sputum in such cases may be apparently negative, although microscopic examination of the affected lung may show active tuberculosis and open tuberculous cavities. The patients described in this paragraph are usually harassed by violent and frequently repeated attacks of coughing with the production of variable amounts of sputum which is forced past even narrow undilatable strictures by the tremendous pressure produced by the act of coughing. Fever is characteristic. In some cases fever occurs daily, while in others it occurs only during periods of excessive retention of secretions, such as tend to occur in women just before or during menstruation. In such cases as these thoracoplasty, by impairment of the expectorating mechanism and perhaps by an increase in the already tight bronchial stricture from even a slight further narrowing or kinking of the bronchus, is likely to increase the toxicity of the patient and cause death.

Although the indications for pneumonectomy given in the last paragraph may be met, this operation cannot be safely performed if there is even a small area of active tuberculosis in the contralateral lung or if the tuberculous bronchial lesions are ulcerative and progressive.

Relatively few patients having far-advanced pulmonary tuberculosis and tuberculous bronchitis present the indications for pneumonectomy that have just been given and at the same time are free from the contraindications. Those patients who have ulcerative, progressive bronchial lesions cannot safely have either pneumonectomy or thoracoplasty unless the bronchial lesions have ceased being progressive. Those patients who have varying degrees of active (although not actively progressive) parenchymal tuberculosis in the less extensively diseased lung that would contraindicate a pneumonectomy may have a thoracoplasty for the more extensively diseased lung. Those patients who have an undilatable bronchial stricture with violent attacks of coughing and with continuous or frequently recurring periods of toxicity from the retention of toxic secretions by the strictured bronchus cannot safely have a thoracoplasty, even if pneumonectomy should be contraindicated, as, for example, by active tuberculous lesions in the other lung.

In our opinion there are, therefore, certain patients for whom only pneumonectomy may be performed, certain patients for whom only thoracoplasty may be performed, and certain patients for whom neither operation can be performed with reasonable safety and hope of a favorable result. In addition, there is a relatively large group of patients for whom pneumonectomy or thoracoplasty may be used but for whom, in our opinion, thoracoplasty is both safer and more likely to produce a good result.



A. *B.*

Fig. 1.—Atelectasis of right lung; great displacement of mediastinum to right; extensive cavitation in upper half of lung. Arrows indicate lateral and superior limits of anterior mediastinal herniation of left lung. Lumen of stenosed (but not ulcerated) proximal main bronchus less than 2 mm. The second roentgenogram (*B*) was made fifteen months after a four-stage posterolateral, and a one-stage anterior, thoracoplasty. Cavities are closed; sputa, negative. All but a part of the anterior mediastinal hernia has been reduced. The patient was referred by Dr. George C. Stucky, Lansing, Michigan.

The particular objective of thoracoplasty is to close tuberculous cavities and to collapse thoroughly all suppurating parenchymal tissue in order to stop the formation of infectious secretions before the bronchial stricture becomes so narrow that secretions cannot be forced past it.

We question the validity of the statement that most so-called atelectatic lungs have reached the maximal degree of collapse and that thoracoplasty, therefore, could produce no further collapse. Not only do many "atelectatic lungs" contain scattered areas of pneumatic pulmonary tissue that are collapsible, but we are convinced that the atelectatic areas themselves become still more compact as a result of thoracoplastic collapse and that tuberculous cavities surrounded by atelectatic lung can often be closed completely by a thorough thoracoplasty. We believe, furthermore, that the removal of the rigid costal wall over an atelectatic lung tends to prevent the development of bronchiectases and new tuberculous cavities by releasing the tension upon the lung.

We have usually been pleasantly surprised by the ability of the great majority of patients to evacuate their secretions by persistent voluntary coughing, even though the lumen of the bronchial stricture is as narrow as 2 or 3 mm. Although we have in many cases repeatedly dilated bronchial strictures before beginning a thoracoplasty, in only two or three of our patients was dilatation of the stricture necessary between the stages of a thoracoplasty in order to release retained secretions. We do not intend to imply by these statements that expectoration is even approximately as simple an act as it is in patients having bronchi of normal caliber. A great majority of patients with stenosing bronchitis have varying degrees of difficulty in expectorating both before thoracoplasty and, especially, during the first few days after each thoracoplasty stage. Since prolonged retention of sputum is a grave danger, meticulous attention must be paid by the surgeon and nurses to the well-known measures that aid the patient in expectorating and, in order to reduce to a reasonable minimum the amount of paradoxical respiratory movement (which is an important cause of difficult expectoration), the total length of ribs resected at each operative stage should be less than if expectoration were easy. Inhalation of oxygen should be used promptly if the patient becomes dyspneic or if the pulse is unduly rapid.

Although some patients with bronchial stenosis (with or without atelectasis) have no cavities or only small ones that are, perhaps, invisible roentgenologically, large cavities are very frequent and "mammoth" cavities are probably more frequently met than in tuberculosis uncomplicated by bronchial tuberculosis. Such large cavities are usually of the balloon type, a greater-than-atmospheric pressure within them being maintained by a check-valve mechanism in the strictured bronchus. Frequently such cavities occur in patients without bronchoscopically visible bronchial tuberculosis and stenosis, the stenosis that has produced the balloon cavity being in a bronchoscopically invisible small bronchus that leads to the cavity. Obviously, one must not assume that all large cavities are of the tension type; they are not likely to be if they never contain a fluid level and if bronchoscopy shows no tuberculous bronchitis.

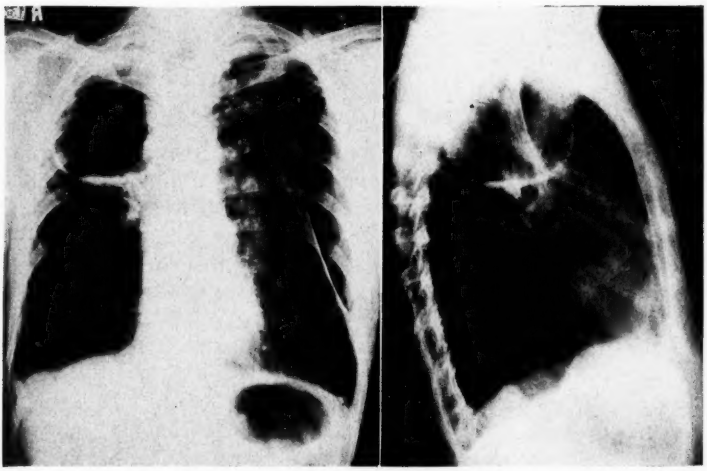
Presumably, large cavities of the tension type should be very difficult to close by thoracoplasty. Actually, we have found that a large majority of such cavities, when they are in the upper third of the lung, close completely. We are unable to offer an adequate explanation of this phenomenon, but we suppose that the great pulmonary collapse produced by the modern type of thoracoplasty overcomes the check-valve mechanism of the bronchial stenosis and that in many cases the walls of balloon cavities are relatively thin and yielding.

We have, on the contrary, been obliged to perform open surgical drainage of a number of cavities in the course of our seven years of experience with thoracoplasty in the presence of bronchial stricture

(in two cases in the five-year, thirty-eight-case series reported in this article). Drainage was required in these cases because the cavities failed to yield to thoracoplastic collapse or because thoracoplasty decreased the already narrow lumen of the draining bronchus, causing retention of toxic secretions in spite of attempts to maintain drainage by bronchoscopic means. In one patient, drainage of a huge cavity was carried out before the thoracoplasty was begun because of toxic symptoms from retention of secretions. When the thoracoplastic collapse over drained cavities is great (or is made so by continuation of the thoracoplasty after drainage), the majority of such cavities gradually become completely obliterated.

A case of a large balloon cavity in the right upper lung deserves special mention because of the peculiar circumstances that caused the death of the patient. The cavity had increased considerably in size during the two months before thoracoplasty and contained a short fluid level. Bronchoscopy showed some scarring of the right upper lobe orifice. Although a small pneumothorax was present on the left side, there was no dyspnea. At the first stage of the thoracoplasty, all the first rib and the second rib to within 4 cm. of its cartilage were resected and a small apicolysis, depressing the apex only to the level of the posterior second rib, was done. Before operation the blood pressure was 90/60; the pulse, 130; and the respirations, 20. During the course of the operation they were, respectively, 130/70, 104, 30, and 90/?, 94, 40. Immediately after operation the blood pressure was 85/65; the cardiac rate, 96 and regular, and the pulse, scarcely palpable. Soon, neither the blood pressure nor the pulse could be detected. There was no dyspnea until shortly before death. The skin was pale, cold, and moist. The patient was conscious and perfectly rational and had a premonition of death. Fifty minutes after operation he suddenly gasped, became unconscious, and died an hour and a half later. The following therapeutic measures were used: intravenous glucose and, later, acacia solution, inhalation of oxygen and helium under slight positive pressure, intravenous coramine, Drinker respirator, and intracardiac injection of adrenalin. When, at the post-mortem examination, the 10 cm. cavity was opened, there was a "gush of wind owing to encapsulated air under pressure." The main bronchus of the right upper lobe was not definitely stenosed; its branches were distributed over the entire surface of the cavity and were traceable to within 1 or 2 mm. of the cavity proper, but no definite bronchial communication could be found. We believe a very narrow bronchial communication with the cavity of the one-way valve type almost certainly existed, even though it could not be grossly demonstrated in the collapsed lung specimen. Death was apparently due to cardiocirculatory failure from the pressure of the completely uncollapsible cavity upon the heart and great mediastinal vessels, even though the pressure was produced by the removal of only two ribs.

Thoracic surgeons have for years recognized that similar symptoms may be produced by even a small stage of a thoracoplasty operation performed in the presence of a pneumothorax or of pleural fluid, the pressure of which has not been reduced by aspiration before, during, or immediately after operation. We believe that, if the pressure of the air in the cavity of the patient being considered had been reduced during or shortly after operation by needle aspiration, cardiocirculatory collapse and death would have been prevented. Since we had never seen or heard of a similar case, the cause of the symptoms and their relief by aspiration did not occur to us in time. Except for those rare cavities that require surgical drainage because of toxicity from retained secretions, all other balloon or tension cavities in our experience have been gradually deflated completely or incompletely by the stages of a thoracoplasty, in spite of the presumed presence of a one-way bronchial valve mechanism.



A. *B.*

Fig. 2.—Large tension cavity with very tightly strictured bronchus. Death occurred from cardiocirculatory failure 2.5 hours after the resection of the first and second ribs because the air in the cavity could not be evacuated by the bronchus, with the result that the pressure produced by the thoracoplasty collapse was transmitted by the tense cavity to the mediastinum. See text for details of this case. The patient was referred by Dr. Samuel H. Watson, Tucson, Arizona.

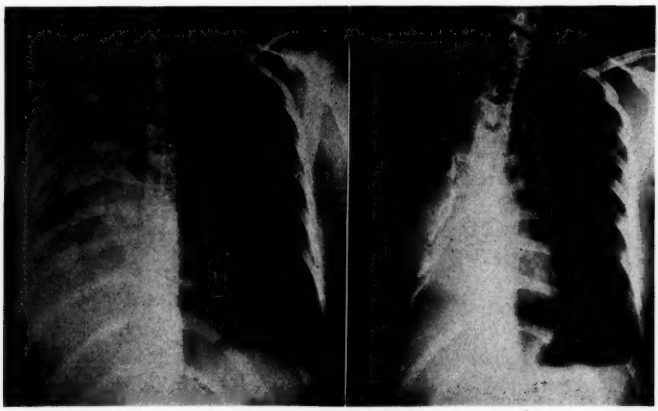
The large cavities, the partial or complete atelectasis, and the extensive pulmonary fibrosis that are present in many cases having bronchial stricture require, for good results, that the thoracoplastic collapse be maximal. Incomplete collapse of the sort produced before the modern thoracoplasty was devised must be blamed for many disappointing results. Maximal collapse in the horizontal plane requires, among many other important things, one of the efficient types of anterior thoracoplasty operation, as well as complete removal of the vertebral trans-

verse processes. When an entire lung is atelectatic, vertical collapse should extend from the apex to the base of the lung, even though a cavity exists only in the upper part of the lung, since an uncollapsed atelectatic part of a tuberculous lung is likely to develop bronchiectasis and tuberculous excavation. Some patients with an extensive stenosis of the proximal part of a stem bronchus have atelectasis of only the upper or the lower part of the lung; this phenomenon is probably due to the fact that the bronchoscopically visible stenosis is insufficiently complete to produce atelectasis and that a greater, but bronchoscopically invisible, stenosis of a lobar bronchus has produced atelectasis of only a part of the lung. If, in such a case, the lower, nonatelectatic part of the lung is roentgenographically not involved by tuberculous infiltration, the surgeon is tempted not to collapse this part of the lung. We believe, however, that it should be collapsed in cases with a high grade of stricture of the stem bronchus because obstructive emphysema and, later, atelectasis with complicating bronchiectasis and tuberculous excavation are likely to develop in this part of the lung as a result of kinking or further narrowing of the stricture from thoracoplastic collapse, or from a continuing activity of the tuberculous bronchitis. In the series of cases we are reporting, atelectasis of the lower part of the lung occurred once during the course of a thoracoplasty and once a month or so after the completion of an upper thoracoplasty, requiring an extension of the thoracoplasty to the base in both cases.

In the cases under consideration in this article, a greater interval between the stages of a thoracoplasty than the customary three weeks is more often required than in uncomplicated cases. The delay may be occasioned by prolonged postoperative fever from retention of secretions, or by a spread of the tuberculosis in either lung. In our present thirty-eight-case series, however, delay between stages was necessary in only three patients (in only one owing to a spread of the disease). In five patients secondary operations were needed for the removal of regenerated ribs in an effort to produce adequate pulmonary collapse; regeneration occurred before the completion of the thoracoplasty either because of long intervals between stages or because of the many stages required in certain cases.

In a large majority of patients the type of operative management that has been outlined results in complete cavity closure as determined by serial stereoscopic Potter-Bucky x-rays. Some patients whose cavities are apparently closed continue to have positive sputum. The source of such sputum is usually in bronchoscopically visible or invisible bronchial ulcers. Twelve of our thirty-eight patients had positive sputum after thoracoplasty; two of these twelve had visible bronchial ulceration and residual pulmonary cavities (the only ones in the series), and both died; six of the twelve had apparently closed cavities but persisting visible ulceration; and four had apparently closed cavities and no visible bronchial ulceration (the sputa of three of these four patients were

positive only on culture). We believe that bronchial ulceration not rarely persists distal to a bronchoscopically impassable fibrous stricture that formerly may have been seen to be ulcerated, the visible ulcer at the site of stricture having healed spontaneously or following treatment with silver nitrate solution or cautery. This is illustrated by one of the patients of this series who, some months after the completion of thoracoplasty and cavity closure, developed a bronchoesophageal fistula that presumably resulted from progression of an invisible bronchial ulcer through the esophageal wall. In certain cases invisible tuberculous ulceration exists in bronchiectases.



A. *B.*

Fig. 3.—The patient made satisfactory progress during four years (1935 to 1939) of pneumothorax; no cavities and negative sputum. In 1935 there was a short period of wheezing, and roentgenograms showed beginning atelectasis at the right base, which became more extensive in 1936. In the spring of 1939 there was a recurrence of wheezing, and roentgenograms showed atelectasis of the right lower lobe and of part of the right upper lobe. Shortly after the abandonment of the pneumothorax in the autumn of 1939 the entire lung became atelectatic, with the rapid development of multiple cavities and displacement of the mediastinum to the right (*A*). Bronchoscopy, March, 1940, showed submucosal infiltration of right stem bronchus and lowermost trachea but no ulceration. Ten-rib thoracoplasty, including anterior stage, produced maximal pulmonary collapse. There were apparent complete closure of all cavities, replacement of mediastinum, and virtually straight spine. Bronchoscopy, December, 1940, showed 2 mm. stenosis of the proximal right stem bronchus without ulceration. During the year following the thoracoplasty numerous concentrated sputum specimens and several cultures have been negative for tubercle bacilli, but, for a period of five weeks ending six weeks ago, one culture and several smears revealed one or two tubercle bacilli after a twenty-minute search. The patient was referred by Dr. Samuel H. Watson, Tucson, Arizona.

Apart from failure to close parenchymal cavities and to render the sputa negative for tubercle bacilli, thoracoplasty may fail to effect complete healing of the tuberculosis because of a spread of the parenchymal disease in either lung which, if it occurs before the completion of the thoracoplasty, may prevent its completion. Dyspnea is another important cause of noncompletion of the operations. Bronchiectasis develops in some, but not all, cases as a result of bronchial stricture; following thoracoplasty the bronchiectases produce a variable amount of purulent sputum, which may be negative or positive for tubercle bacilli. We recently performed a lower lobe lobectomy on a patient who had no

bronchial stricture and whose upper lobe tuberculosis was apparently completely healed, the positive sputum presumably arising in the bronchiectatic lobe; on section of the lobe, tuberculous ulcers were found in the walls of the bronchiectases; the scanty sputum was negative after the lobectomy. A relatively rare cause of failure of thoracoplasty in the group of cases that are the subject of this article is persisting toxicity resulting from unhealed caseous foci in the collapsed lung and from puddled secretions in the bronchial tree that the patient is unable to expectorate efficiently. Relapse to tuberculosis activity or the reappearance of positive sputum months after an apparently excellent result has been obtained from thoracoplasty occurs more often in patients with high-grade bronchial stenosis, especially when bronchial ulceration exists, than in patients without bronchial complications.

Pneumothorax has been disappointing when used for pulmonary tuberculosis complicated by bronchial stricture in a large majority of the patients we have seen in consultation in various sanatoriums. We have, however, seen a number of patients whose condition was entirely satisfactory while an adequate pneumothorax was being maintained but in whom positive sputum and the syndrome of active tuberculosis with bronchial stricture recurred, or first occurred, when the pneumothorax was abandoned (Fig. 3). Consideration should be given, therefore, to the indefinite maintenance of a pneumothorax that is clinically entirely satisfactory, provided that the considerable danger of the future occurrence of some serious pleural complication be accepted.

Pneumothorax was initially used for a variable time (in a few cases in conjunction with temporary phrenic paralysis) in thirteen of the thirty-eight cases of this thoracoplasty series. Incidentally, we are opposed to the use of phrenic paralysis in the presence of tuberculous bronchitis and, if a temporary phrenic paralysis operation has been performed, we prefer to wait, if feasible, until the function of the diaphragm has returned before beginning a thoracoplasty. Although adhesions that prevented complete pulmonary collapse by pneumothorax were present in most of the cases, a few had a degree of collapse which was great but insufficient to close the cavities in the atelectatic lungs. We are convinced that the modern type of thoracoplasty produces a greater, or at least a different, more constant, and more effective type of collapse of an atelectatic or fibrotic upper lung than does even a positive-pressure pneumothorax without pleural adhesions; the advantage of thoracoplasty in such cases is probably that the pressure upon the lung by the scapula and the temporarily used anterior brace is greater and more constant than that of pneumothorax air.

In twenty-five of our thirty-eight cases the principal tuberculous tracheobronchial lesion was in the main stem bronchus, the proximal part of the bronchial lesion being at the level of the carina in eight of these twenty-five cases; in five of the twenty-five cases there were also lesions in the trachea. In a few cases the chief bronchoscopically visi-

ble lesion was at the orifice of the right middle lobe bronchus or in a lower lobe bronchus. In a few cases there were small and, at the time of thoracoplasty, apparently indolent lesions in the bronchi of the less extensively diseased lung.

In the first part of this article it was reported that, of our thirty-eight patients, three (7.9 per cent) are dead, ten (26.3 per cent) have positive sputum (three being positive only by culture), and twenty-five (65.8 per cent) have negative sputum. In eleven of the thirty-eight cases having a bronchial stenosis with a lumen of from 4 to 6 mm. without visible ulceration, the sputum became negative in nine and remained positive in two after thoracoplasty. In nine cases with a 1 to 3 mm. stenosis without visible ulceration, the sputum became negative in six and remained positive in three. In three cases with a 4 to 6 mm. stenosis with ulceration at the time thoracoplasty was begun, the sputum became negative in one and remained positive in two. In six cases with a 1 to 3 mm. stenosis with ulceration, the sputum became negative in two and remained positive in four. In nine cases with relatively acute tuberculous bronchitis (seven having ulcerative lesions and two having severe inflammatory lesions) the sputum became negative in seven and remained positive in two. Among the patients listed here as having positive sputum are included the three dead patients, although one of them had a closed cavity and negative sputum upon completion of the thoracoplasty; a progressive ulcerative bronchitis developed later and led to death.

These figures indicate, in spite of the small number of the cases in the series, that the best results may be expected in the acute bronchitis cases and in those not having visible ulceration, even when there is a moderate or high grade of stenosis. The behavior of tuberculous tracheobronchial lesions is, in the present state of knowledge, mysterious and unpredictable. We do not know under what circumstances they arise or become healed. Many ulcers heal rather promptly under treatment with silver nitrate; some fail to do so; and others heal without treatment. Although we have occasionally seen bronchial lesions first appear during the course of a staged thoracoplasty, or after its completion with cavity closure, we believe that the pulmonary collapse was not the cause of the bronchial lesions. Our experience with thoracoplasty in the presence of tuberculous tracheobronchitis, which now includes approximately sixty cases, has not led us to believe that complete control of the parenchymal lesions by adequate collapse is necessarily followed by anything like a corresponding improvement in the tracheobronchial lesions, and we are almost of the opinion that control of the parenchymal lesions has no effect, good or bad, upon the highly important ulcerative tracheobronchial lesions. Too few statistical data are as yet available definitely to confirm or disprove this tentative opinion.

A consideration of lobectomy was omitted from the first part of this article in which the relative advantages and disadvantages of pneumonectomy and thoracoplasty were discussed because the indications for, and contraindications to, lobectomy are in most respects different from those of pneumonectomy. Although the symptoms and physical conditions calling for a total pneumonectomy can occur with regard to a single lobe, they are rare since bronchial lesions producing such a state of affairs almost constantly involve the main stem bronchus as well as lobar bronchi. The greatest usefulness of lobectomy is probably for those patients with tuberculous parenchymal lesions (with or without tuberculous bronchitis) which are strictly confined to a lower, or to the right middle, lobe and are unlikely to become completely healed as a result of thoracoplasty and for which a thoracoplasty, if undertaken, would need to include the uppermost ten or eleven ribs, thereby sacrificing the function of a healthy upper lobe. Such lesions include (1) relatively large cavities lying close to the diaphragm or in the lower half of the costovertebral gutter and (2) bronchiectases that alone are the source of tubercle bacilli in the sputum (or, in negative-sputum cases, that produce disabling symptoms). We believe that lobectomy should not be used even for large cavities in the upper lobe because the far less dangerous operation of thoracoplasty completely closes such cavities in a large majority of cases. Bronchiectasis confined to an upper lobe in itself rarely requires either lobectomy or thoracoplasty.

Lobectomy is being increasingly discussed as the operation of choice for those patients in whom maximal thoracoplasty collapse has failed to close a tuberculous cavity completely and in whom all the other tuberculous lesions in the lung have apparently become well healed. We have not used lobectomy for this indication as we believe that direct surgical drainage of the residual cavity offers a much lower operative and postoperative mortality and that, in a considerable majority of the cases, the sputum will become negative and the cavity will eventually become obliterated as a result of the growth of granulation tissue and epithelium.

What we have written in this article clearly indicates that we believe the indications for pneumonectomy or lobectomy in tuberculosis are rare. In our extensive experience with the surgical treatment of this disease, we have performed the former operation only twice and the latter three times. One pneumonectomy patient, who presented the definite indications for the operation given in the early part of this article, is living five years after operation and is at present disabled by an exquisitely tender tuberculous wound. The other pneumonectomy patient, who was toxic because of the retention of nontuberculous secretions in a lung that had been well collapsed by a total thoracoplasty and phrenic paralysis, died thirteen days after operation from the rupture of a necrotic area of the aorta. The walls of an indolent tuber-

culous empyema cavity, without a bronchopleural fistula, were removed with the lung. Retention of pulmonary secretions in this patient was due to the loss of an efficient expectorating mechanism as a result of the complete thoracoplasty and phrenic paralysis and was not due to bronchial stricture. The three lobectomy patients are well. One had a densely fibrotic, tuberculous middle lobe with a cavity that had resisted closure by a good pneumothorax and phrenic paralysis. One had a basal bronchiectasis, but the additional tuberculous infection was not recognized until after the lobectomy operation, when an acute tuberculous pneumonia appeared in the contralateral lung and gradually cleared completely. The third patient had healed tuberculous lesions in the upper lungs but persistingly positive sputum that presumably came from the bronchiectatic lobe; on section this lobe showed tuberculous ulcers in the bronchiectases; the sputum became negative after operation.

SUMMARY

1. There are, in our opinion, certain clear-cut indications for, and contraindications to, both pneumonectomy and thoracoplasty in the treatment of pulmonary tuberculosis with bronchial stricture, atelectasis, and fibrosis that must be strictly observed if a high percentage of therapeutic failure is to be avoided.

2. In some cases neither pneumonectomy nor thoracoplasty can be used safely.

3. For those particular cases in which no contraindication exists to either pneumonectomy or thoracoplasty, we believe thoracoplasty is the better operation because the mortality is much lower and the percentage of apparent healing of the tuberculosis is much higher.

4. The factors that influence the success of thoracoplasty in the group of complicated cases under consideration, as well as those factors responsible for failure, are considered. The results in a series of thirty-eight patients operated upon during a five-year (1934-1939) period are given.

5. The indications for lobectomy are discussed.

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DISCUSSION ON "TREATMENT OF ENDOBRONCHIAL TUBERCULOSIS" BY DR. J. MAXWELL CHAMBERLAIN AND DR. JOSEPH GORDON;* "TUBERCULOUS STENOSIS OF THE MAJOR BRONCHI" BY DR. WILLIAM M. TUTTLE, DR. E. J. O'BRIEN, DR. J. C. DAY, AND DR. F. J. PHILLIPS;† AND "EFFECT OF THORACOPLASTY UPON PULMONARY TUBERCULOSIS COMPLICATED BY STENOTIC TUBERCULOUS BRONCHITIS" BY DR. JOHN ALEXANDER, DR. GEORGE N. J. SOMMER, JR., AND DR. ADRIAN A. EHLER

DR. EDWARD J. O'BRIEN, Detroit, Mich.—I believe it must be obvious to all of us that we are discussing an extremely grave condition.

In our clinic and in others which have reported, results are uniformly bad with artificial pneumothorax in bronchial stenosis and have been fairly good—not excellent (because they could not be excellent with anything)—following thoracoplasty.

Because of the bad results in ulcerostenosis following pneumothorax, I believe we should seriously consider bronchoscoping all patients before instituting it, as we now do, routinely, before thoracoplasty.

I believe this should be done, especially if there is retraction of the trachea and mediastinum, with some suggestion of atelectasis. To institute a pneumothorax under these circumstances without a preliminary bronchoscopy, especially if there are wheezing and other symptoms that accompany a stenosis, is inviting trouble. By doing this, I am sure we can prevent many of the catastrophes which have occurred.

The question of pneumonectomy, in patients with stenosis and blocked cavity, always presents itself. The results from this procedure, in tuberculous ulcerostenosis, have not been very good.

When, however, thoracoplasty appears not to be the procedure of choice because of definite blocked cavity (and naturally no form of collapse is going to close a blocked cavity), we must, I believe, make a decision between three procedures—pneumonectomy, cavernostomy, or cavity drainage plus thoracoplasty. Some good results have been obtained from each.

I do not believe anybody has enough experience to know which, eventually, is going to be the safest procedure. I do not know; if somebody does, I wish he would tell me.

*See page 292 for article by Chamberlain and Gordon.
†See page 299 for article by Tuttle, O'Brien, Day, and Phillips.

DR. J. D. STEELE, Milwaukee, Wis.—I believe it should be pointed out that the obstruction produced by a fibrous stenosis of the tracheobronchial tree is not always permanent. Over a period of months, some of these definite fibrous stenoses will thin out and dilate spontaneously. Irreparable damage may be done in the meantime. Sometimes it is possible to keep a stenosis dilated and wait for spontaneous dilatation to take place.

I am glad to see attention given to flaccidity of the bronchial wall as producing one form of bronchial obstruction. It certainly is a very important form of bronchial obstruction and, as far as I know, has received no proper attention in the literature up to this time. Patients having this type of lesion must be educated as to coughing. If they cough gently, the sputum will come up much more easily than if they cough forcibly. Of course, the forcible cough merely increases the bronchial obstruction.

These lesions always tend to disappear spontaneously. I believe fibrous tissue is laid down where the cartilage has been destroyed.

DR. ETHAN FLAGG BUTLER, Ithaca, N. Y.: I wish to compliment the essayists on their able presentation of this important topic.

My discussion will be limited to the subject of pneumonectomy in the presence of, and primarily because of, tuberculous bronchitis. The prerequisites for pneumonectomy under these circumstances must be extremely rigid. The surgeon must be convinced by every reasonable diagnostic measure that all present active tuberculosis is limited to the lung which is to be excised. The surgeon must likewise be satisfied that the present condition of the bronchus will permit of primary healing of the bronchial stump. The pleura must be uninvolved. The contralateral lung should have been subjected to an adequate test of its ability to remain free of tuberculosis while carrying the full burden of respiratory function. Obviously, not many will satisfy these requirements—probably less than 0.1 per cent of all patients with pulmonary tuberculosis.

If we accept the dictum that tuberculosis is more easily controlled when lung volume is diminished, may we not also with equal propriety feel that tuberculosis will be more difficult to control when lung volume is increased? Therefore, homolateral thoracoplasty to prevent mediastinal shift and consequent overexpansion of the contralateral lung might well follow every pneumonectomy for pulmonary tuberculosis.

Whether or not pneumonectomy for pulmonary tuberculosis wins an accepted place, only time can tell. This much, however, has been proved, that the bronchi will heal and that these operations can be done without inevitable occurrence of bronchial fistula or tuberculous empyema.

In 1940 I reported such a pneumonectomy. Further remarks on that patient are now in order. Her surgical convalescence was uneventful. Four months after pneumonectomy, however, she developed reactivation at the opposite apex. Under rigid medical supervision, she barely held the lesion in check. Ten months after pneumonectomy a thoracoplasty of the obsolete paravertebral type was done in one stage. This allowed her mediastinum to return to its midline position. Since the thoracoplasty on the pneumonectomy side, her sputum, which had been positive on direct smear, has become negative on concentration. It is too early to report negative cultures on her sputum or negative gastric lavage. The lesion at the opposite apex is much smaller and definitely "harder" in its roentgenologic appearance.

I wish to place briefly on record a second pneumonectomy done primarily for active tuberculous bronchitis, done in the presence of a smear positive sputum, done in the presence of a therapeutic pneumothorax of many months' duration, done only after all evidence of active tuberculosis elsewhere had been carefully excluded.

The patient, at the time of operation, was a married Italian woman, aged 23 years. She had had pulmonary tuberculosis for approximately 3.5 years. At onset there was bilateral involvement. She was ultimately able to stabilize the disease on her right side. She was admitted to the Mount Morris Tuberculosis Hospital two years ago, at which time she was six months pregnant. The baby was delivered by cesarean section at term. One month after that operation and nineteen months before pneumonectomy, therapeutic pneumothorax was successfully induced on the left side and was maintained up to the time of pneumonectomy. Obliterative pleuritis caused some re-expansion, but there were no other pleural complications.

Thirteen months before pneumonectomy bronchoscopic examination disclosed normal bronchi on both sides. Six and a half months before pneumonectomy active tuberculous bronchitis, involving the left lower lobe bronchi, was demonstrated. The evolution of this complication was carefully followed. It never involved the left main bronchus nor did it involve the upper lobe bronchi. It remained limited to the left lower lobe bronchi which showed, first, typical tuberculous granulation tissue, and later, during the regressive phase, typical fibrostenotic changes. At every examination abundant purulent secretions could be aspirated from the lower lobe bronchi. These secretions and the patient's sputum were always strongly positive for tubercle bacilli on direct smear.

Pneumonectomy was done on April 3, 1941. Convalescence was uneventful. The pleural cavity was not drained, but was allowed to fill with sterile effusion. Six weeks after pneumonectomy an old-fashioned paravertebral thoracoplasty was done in one stage on the pneumonectomy side, in order to prevent mediastinal shift and consequent overexpansion of the opposite lung. Convalescence from the thoracoplasty was uneventful. The wound is well healed. The patient is free of any complications, but she is still on a rigid bed rest program comparable to a post-thoracoplasty program. Since pneumonectomy, three sputum specimens have been examined. All three were negative on direct smear; one was negative on concentration.

DR. FRANK S. DOLLEY, Los Angeles, Calif.—Endobronchial tuberculosis, because of interference with bronchopulmonary drainage from involved areas distal to it, is a real menace to every patient who has pulmonary tuberculous disease.

Artificial pneumothorax as a curative procedure in the treatment of marked or progressive stenosis of a main stem or secondary bronchus often produces alarming, even disastrous, results. Retention of infected material, atelectasis with rapidly developing bronchiectasis, or bronchopulmonary abscesses are by no means unusual complications.

If bilateral bronchial involvement is evident endoscopically, or probable symptomatically, surgery is at that time very hazardous, for no one can foretell the end result of this endobronchial disease as regards future bronchial patency.

If disease is unilateral, however, surgical relief is to be considered. If the main stem bronchus is seriously involved, if there is marked stenosis, if progression seems likely that might well result in almost complete atresia, it is my opinion that the removal of that portion of the lung distal to this area, whether it be a lobe or an entire lung, may be required to effect definite arrest. I feel this to be likely even though but little parenchymatous involvement may be evident in the lung distal to the bronchial area affected.

On the other hand, amazingly satisfactory results not infrequently follow thoracoplasty. In the great majority of cases this operation should be given a trial. Lobectomy or pneumonectomy is not precluded if thoracoplasty proves unsuccessful.

If, however, marked lung destruction with bronchial distortion and puddling exists, thoracoplasty will not improve the condition. The situation is one of extensive bronchopulmonary suppuration and should be so treated—by the removal of the lobe or lobes involved.

DR. LEO ELOESSER, San Francisco, Calif.—It seems that the indications for thoracoplasty in bronchial stenosis are not yet as precise as we would wish to have them, and I do not know how to make them any more precise. It is doubtless true that we have done in the past, particularly in the early days, many thoracoplasties for stenosis without knowing what we were doing, and that many of these people got well. I believe in the main that, if the lung is carnified, and if in this carnified lung there are bronchiectatic cavities, we cannot expect too much from thoracoplasty.

I believe that drainage of cavities in stenotic lungs should be reserved for real cavities, because to attempt to drain bronchi in bronchiectatic lungs is disastrous.

DR. WILLIAM M. TUTTLE.—Dr. Steele stated earlier this evening that he felt that in certain cases of fibrous stenosis dilatation occurred spontaneously. I do not believe that a true fibrous stenosis ever dilates in this manner. We have seen at times what we felt were fibrous stenoses and have described them in our operative notes. Later we have seen these narrowed lumens return almost to normal. I cannot help but think that we were mistaken in our first appraisal and that that which we had thought to be a fibrostenosis was in truth a very localized type of induration and edema which in time disappeared and left a normal bronchus. This view I think is further borne out by the general tendency of fibrous tissue to shrink. By such a token the bronchial lumen would become smaller, rather than assume its normal size. I believe I can safely say that in no instance have we ever seen a true fibrous stenosis disappear, but in numerous instances we have observed a moderate stenosis become very marked with the passage of time.

Naturally, the discussion tonight is centered about the relative merits of pneumonectomy versus thoracoplasty as proper methods of treatment in those cases of bronchial stenosis with extensive poststenotic parenchymal disease. There is one point which must be borne in mind and that is that in many patients bronchial disease is present without evidence of parenchymal tuberculosis. It is only after some time that sufficient spilling from the bronchus occurs to infect the parenchyma. In such patients, the infection in the bronchus invades the deeper layers and any violation of this territory, even in the face of an apparently healed lesion, would undoubtedly free viable tubercle bacilli which would certainly be a menace to proper bronchial healing.

It has been suggested that chemotherapy might have value in the treatment of the suppurative process which often takes place in the pulmonary parenchyma distal to the stenosis. Earlier today I stated that we had tried such treatment in a respectable and representative number of patients with poor results. Such response is in line with our findings that the organism involved in poststenotic suppuration is in general an anaerobic *Streptococcus viridans*. Chemotherapy has not given encouraging results when used against anaerobic bacteria.

DR. JOHN ALEXANDER.—Persisting toxicity from the retention of pulmonary secretions by an undilatable bronchial stricture is, I believe, the most important symptom demanding consideration of pneumonectomy. Although a tuberculous pulmonary cavity may be present in such cases, the absence of roentgenologically visible cavitation is not rare.

Dr. Butler has made the important point that the diffuse white roentgenological shadow produced by a completely atelectatic, fibrotic lung does not exclude the presence of a complicating empyema that may not have been suspected or that may not have been reached by diagnostic thoracentesis. Dr. Haight and I have each found at the time of an intended pneumonectomy for tuberculosis extensive extrapleural pockets of pus that prevented the reasonably safe freeing and removal of the lung. In a recent patient, who had had a thoracoplasty, I unexpectedly found a long narrow empyema cavity filled with pus and I was able to remove the

lung and the walls of the empyema together; this patient did well after operation until she died suddenly on the thirteenth postoperative day from the rupture of a small necrotic area of the mid-thoracic aorta.

Dr. Butler may be right in believing that a pneumonectomy should be performed in the early stages of unilateral tuberculous bronchial disease, before atelectasis and extensive destruction of the lung have occurred, but I doubt it. I believe the risk of pneumonectomy in tuberculosis is too great for this use of the operation when a large proportion of patients with early tuberculous bronchial lesions recover with only bronchoscopic treatment and, perhaps, some minor collapse therapy operation, and when a majority of those with bronchial stricture and severe parenchymal tuberculosis recover as a result of thoracoplasty.

In his discussion at last year's meeting, Dr. Eloesser was rather scornful of thoracoplasty in the presence of bronchial stricture. I am happy to hear that this year he seems more than a little receptive to it.

EXTRAPERIOSTEAL PNEUMONOLYSIS IN PULMONARY TUBERCULOSIS

A Preliminary Report

Charles P. Bailey, M.D. (by invitation)
Philadelphia, Pa.

THIS collapse procedure represents an attempt to overcome the more serious complications of both extrapleural pneumothorax and ordinary staged thoracoplasty. It has always seemed to me that the phenomena of paradoxical breathing, mediastinal flutter, atelectasis, and bronchiogenic spread, following thoracoplasty, were due mainly to loss of rigidity of the chest wall and to the resultant impairment of cough function. These symptoms are conspicuously absent in cases of extrapleural pneumothorax, even though the amount of produced pulmonary collapse is much greater. On the other hand, the tendency to ischemic necrosis of the outer wall of collapsed cavities and the danger of early or late uncontrollable re-expansion of the lung in extrapleural pneumothorax make this a none too satisfactory procedure.

Churchill's method of utilizing the deperiostealized ribs to prevent paradoxical breathing in the two-stage procedure of pneumonostomy for lung abscess in small children, Semb's method of mobilizing the pulmonary apex, Eloesser's subcostal lung compression, and Alexander's experience with supraperiosteal packing for pulmonary cavities, and his observation of the slight chance of cavity perforation in these cases have been freely drawn upon in devising this operation.

TECHNIQUE

The usual thoracoplasty incision is made, and the estimated number of ribs are completely denuded of periosteum from transverse processes nearly to the costal cartilages. A 2.5 inch piece of the second rib is resected, and the periosteum on the under surface of the first rib is removed. A very extensive Semb apicolysis is performed, freeing the apex down to the hilum, and continuing all the way down the costovertebral gutter to the lowest denuded rib. The intercostal muscles, nerves, and vessels are cut posteriorly so that the lung is completely mobilized out of the gutter. The anterior attachments of the intercostals are left intact, thereby maintaining a rich blood supply from the internal mammary arteries. This leaves a portion of lung tissue adherent anteriorly, so that Dr. Louis R. Davidson's modification of the Monaldi method of intracavitary catheter drainage may be employed safely in cases of check-valve cavity. The wound is closed in the usual thoracoplasty manner.

The space is maintained by aspirations and refills much like an extrapleural pneumothorax. The collapse is routinely a very good anatomical or apical selective

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type. After three to six months, there is practically complete bone regeneration on the lung surface, rendering the collapse permanent.

At any time after two months, the case may be safely and readily converted into a thoracoplasty by removing the denuded ribs. It is questionable whether this second operation can or should be avoided.

RESULTS

In a period of a little over one year, nineteen patients have undergone this operation. No bronchiogenic spread has resulted, and no serious paradoxical breathing has developed, although up to ten ribs have been stripped at one time in patients from 15 to 57 years of age. In several cases the vital capacities were between 1,300 and 1,500 c.c., and three cases had acute exudative febrile disease.

Of the fifteen unilateral cases, eleven are over two months postoperative. One of these died six weeks later as a result of maniacal psychosis, wound rupture, secondary infection, and inanition. The other ten have had cavity closure and sputum conversion. Of the four bilateral cases, one is very recent, one is sputum negative, having had contralateral clearing; one is positive from the other side and is now having pneumothorax therapy; and one patient died of contralateral progression, ten months later.

In one recent case due to extreme fibrosis an apicolysis could not be performed. Seven days later the patient developed a staphylococcic infection in the space, perhaps due to lung injury at the time of the attempted apicolysis or to a breach in the refill technique. No other primary (?) infection of the space has occurred.

The first case operated upon developed a wound rupture on the sixth day after a high positive pressure refill. A secondary infection of the space occurred. High positive pressures are no longer employed.

Three cases have been converted to thoracoplasty to date, up to nine ribs being removed at one time.

Several of the early cases developed superficial wound infections, apparently due to decubitus pressure. This is now avoided by the prone position.

SUMMARY

It is believed that an improved method of surgical collapse has been developed. Further observation will determine its eventual place in tuberculosis therapy.

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DISCUSSION

DR. J. ANTRIM CRELLIN, Philadelphia, Pa.—It has been my opportunity to follow medically the majority of Dr. Bailey's cases. He has operated upon patients with acute and chronic tuberculosis. Some have been good risks, and others, bad risk cases. It would seem to me that in the bad risk cases this operation would be valuable, the same indications being used as on those of extrapleural pneumothorax several years ago.

My observations postoperatively on seven cases which were followed regularly in the dispensary showed no contralateral spread. Paradoxical respiration was minimal, and the amount of collapse is comparable to a selective pneumothorax. Postoperative shock also was comparable to that in patients on whom an extrapleural pneumothorax had been done.

Sputum conversion in the majority of instances occurred quite rapidly, within a period of a month. Certainly we shall have to wait a period of time before we can determine just exactly what the results will be. However, it seems that the cases that have been treated have reacted most favorably.

DR. WILLIAM M. TUTTLE, Detroit, Mich.—We have been impressed by the papers dealing with thoracoplasty and by the excellent results obtained. While one should not deprecate the attempt on the part of any investigator to find a new way to do an old trick better, still it is difficult to see why almost yearly constant attempts are made to invent new and bizarre operative procedures to replace or at least precede thoracoplasty. Especially is this true when it has become repeatedly more obvious to many that thoracoplasty is the best procedure in instances where permanent collapse is desired.

Dr. Bailey has resorted to extraperiosteal pneumonolysis in the hope of obviating paradoxical respiration, postoperative shock, and all those ills which from time to time befall the patient who has undergone thoracoplasty.

It seems to me that such fears do not outweigh the dangers inherent in the procedure which has been described. In the first place shock is not in this day a serious consideration in most instances in which thoracoplasty is used. Certainly, the element of shock present in a first stage thoracoplasty should not be as great as in a procedure which strips from one-half to two-thirds of the lung free from the chest wall. Secondly, paradoxical respiration in our present experience has ceased to be a serious complication. Its dangers have been obviated by more careful attention to lung movement at the time of thoracoplasty, by keeping the airways free from secretions which may block them (in some patients immediate or late bronchoscopy may be needed), and by the postoperative use of a medium weight shot bag over the decostalized area.

The procedure as described is really nothing more than an extrapleural pneumothorax with embellishments. The latter operation in our hands has not been totally satisfactory because the degree of postoperative shock which its use engenders is often great, the number of complications is more, and the good results

are fewer than with thoracoplasty. Furthermore, a large extrapleural space is left which in time must be obliterated; this may not be an easy procedure, especially if tuberculous infection should involve this space.

In one or perhaps two of the patients subjected to extraperiosteal pneumonolysis an extensive stripping has been done to control a relatively small apical lesion, and good basilar lung has been wasted needlessly.

It may seem that I have been overly harsh in this criticism, but I feel strongly that extraperiosteal pneumonolysis is a procedure which has been devised to avert certain theoretical dangers of thoracoplasty and by such good intent has taken to itself dangers far more ominous.

DR. ANTHONY D. CRECCA, Newark, N. J.—I should like to put in a plug for the extraperiosteal operation which Dr. Bailey has described. There is no doubt that in children thoracoplasty is a very deforming procedure, and here you have a procedure, or technique, which will give them a new second bony structure with a fixation of the spinal column before you do the permanent thoracoplasty, thereby avoiding the scoliosis which would otherwise occur.

One little girl, who had a thoracoplasty performed when she was 11, had scoliosis that was so terrific by the time she was 16 that she was permanently deformed.

Secondly, we have used the same technique, as you may have seen, with our plombage procedure that Dr. Dieffenbach showed you. There we performed the usual three-rib thoracoplasty and decollated the lung to the seventh or eighth posterior rib. We filled the space with the hydrosteroline, which you heard described. Under this plombage, regeneration of rib occurs. The control of the lesion occurs immediately, and there is no hurry for doing the second or third stage, because of the too rapid regeneration of bone or lack of control of the lesion.

Recently we performed the same procedure, as described by Dr. Bailey, on a nurse with extensive involvement of one side, with a temperature of 102° F. to 103° F. We used the same technique that Dr. Bailey described except that we left the periosteum on the dorsal portion of the rib to maintain the viability of that rib. We decollated this lung from the first to the tenth posterior ribs. Immediately the temperature went down to about 100° F., respiration and shock symptoms disappeared; and she showed an entirely different picture.

I believe in the acute stages, when the patient is slipping away from you, the procedure may be used as a temporary procedure to control the lesion until the patient is in shape to complete the thoracoplasty.

This procedure may also be used to supplant the use of extrapleural pneumothorax since it removes the danger of bronchopleural fistula to a greater extent.

DR. JOSEPH W. GALE, Madison, Wis.—I believe we already have enough operations in our armamentarium for the treatment of pulmonary tuberculosis. We have used all of them in the past, with good, bad, or indifferent results.

It seems to me, however, that this procedure will combine the dangers of the Semb apicolysis with those of extrapleural pneumothorax. As I understand it, in this operation the muscle bundles and the vessels are divided near the transverse processes, and pleural separation is carried out along the vertebral gutter, separating the lung from the vertebral bodies.

Dr. Oatway and I gave a paper in Atlanta and discussed some of the dangers involved in extrafascial apicolysis. We have encountered plenty of trouble. Several accidents occurred when we were separating the lung from the vertebral bodies. Many tuberculous cavities located in the apex lie posteriorly next to the vertebral bodies. If one forces a separation at this point, he is apt to perforate the cavity, or destroy the blood supply to the cavity wall, so that at some future time a rupture of the cavity may occur.

As far as the extraperiosteal separation is concerned, I believe the author has eliminated the danger of cavities lying posteriorly and peripherally. However, many others lie in the vertebral gutter; therefore, since the separation must be extrapleural, the dangers are still present.

I should prefer to have a tuberculous infection occur following an extrapleural pneumothorax, because it is confined to a closed space, rather than outside the chest wall as it would be in the author's type of operation.

There is one other point which interests me greatly and which concerns the patient upon whom we have performed an extrapleural pneumothorax. If an opening occurs in the line of suture, allowing air to escape into the tissues, extensive emphysema develops very quickly.

I wonder how shortly after the type of operation just described one is able to keep the air confined to the desired space. In these cases in which such a tremendous amount of collapse occurs, if infection supervenes, it seems to me that it will be very widespread and cannot be brought under control except with the greatest difficulty.

DR. RICHARD H. MEADE, JR., Philadelphia, Pa.—Since hearing of Dr. Bailey's procedure, and being considerably impressed by its value in a very limited type of case, I have carried out the procedure, not as he has done it, but according to his general principle, in six patients.

I am thoroughly in agreement with Dr. Tuttle and others that thoracoplasty is, in general, the best procedure. I am still of the opinion, however, that there are certain patients—and I know I see them—who are too sick to stand thoracoplasty, whether one does it by one or more ribs at a stage. They still, some of them, cannot tolerate even that much interference with their breathing.

I believe, therefore, in such desperately poor-risk patients, that this type of operation has a very distinct field of usefulness. I believe, furthermore, that in the sick patient it should be staged. I have just completed the procedure in a very sick patient upon whom I did the operation in three stages. I did not carry it out nearly so extensively as Dr. Bailey has shown.

It gives a form of collapse without deformity, which is of great value, as has been pointed out, in the growing child. I believe that otherwise the overlying rib probably should always be removed, because of that persistent danger of development of infection in the extraperiosteal space, even months after the operation has been completed.

I believe this is another modification of an older operation which may have considerable value or may have very little value, but I do think it is worth a trial.

DR CHARLES P. BAILEY.—Dr. Tuttle feels that thoracoplasty as now performed is very satisfactory and sees no reason for any new operation. From the reports that we have had from five excellent clinics today, you can see that the general average of good results in thoracoplasty is about 70 per cent. It is a counsel of defeat to say that this is the best we can ever have and that we should not try to do any better.

He feels that we sacrifice more lung tissue by this operation than we do with thoracoplasty. I believe that probably we have done more collapse on my cases than necessary. I have always used the rule that we were taught at Sea View Hospital to go well below the lesion on the lowest rib in doing a thoracoplasty. Naturally, since we do this operation all in one stage, we could not do it down to the fifth or sixth rib and then wait for possible cavity closure before doing the seventh or eighth. But I have used the same rule as I would have used in a thoracoplasty. Perhaps one can stop sooner, because we certainly strip farther

forward and out of the costal-vertebral gutter, and, therefore, the effectiveness of the collapse should be greater. Perhaps we can save two or three ribs in the future.

We have done only nineteen of these operations, and we were learning while we did them. Probably we do not yet have the best technique possible. Dr. Tuttle feels that the procedure is no better than extrapleural pneumothorax. The difference between this and the extrapleural pneumothorax is that the intercostals remain on the lung, giving a richer blood supply to the lung surface. Therefore, the danger of perforation of tuberculous cavities in that region is no greater than in thoracoplasty, which is admittedly small.

Next is the thought that there might be trouble in maintaining the space as in cases of extrapleural pneumothorax. Sometimes in extrapleural pneumothorax, the lung will re-expand to a considerable degree during the first few days with loss of an efficient collapse. That has not happened in these cases, except the one. In all cases but one, we have had excessive amounts of fluid which held the space very beautifully. In fact, the danger was rather rupture of the wound than of losing the space. The air leaks out quickly during the first two or three days, and the space is really held by virtue of the fluid.

Once the bone is completely regenerated over the lung surface, loss of space cannot occur, because of the rigid bony walls. This is in contrast to the tendency to late shrinkage of the space in extrapleural pneumothorax.

We have not abandoned thoracoplasty by any means. Maybe, in thoracoplasty, I have been removing a little more rib than some of the men who talked today, and I have had a lot of paradoxical breathing in some cases. Most patients do not die from it, but it is annoying. We do not get much shock in thoracoplasty, and I believe that is fine, but we do not get it in this procedure either. We routinely transfuse these cases postoperatively.

This is really not a modification of extrapleural pneumothorax if one is going to perform the second operation, because that converts it to thoracoplasty. We have only converted three so far; the reason is that we do not know what might happen, and I thought we might learn something by waiting.

These cases do not seem to become infected easily. I have a couple of cases over a year old. In one case I have allowed the space to fill with fluid right to the top, and she has not had any trouble. Maybe she will, and I shall then be very sorry for waiting.

But this is only a preliminary report. In other words, we believe a thoracoplasty is an all-right procedure, but the principle of softening a section of the chest wall with each stage, and doing enough stages to collapse the lung that way, seems to us to be physiologically wrong. If one can collapse the lung at one time and do the rib removal later on, one should never have paradoxical breathing or interference with cough and expectoration.

I have used this operation in one child, a 15-year-old girl, and, of course, she did not get any deformity. If we do the thoracoplasty on her later, I believe the deformity will be less than with an initial thoracoplasty, as the new regenerated bone plate should help prevent scoliosis.

Dr. Gale said we have too many operations, but there is always room for a good one. If this is not a good one, it will soon be discovered.

He also touched upon a very important point, one that I have considered, namely, the separation of the lung from the upper mediastinum and the costal-vertebral gutter. It is true there is no periosteum on the lung apex, and the danger of perforation of a cavity in that region would be equivalent to that in that portion of an extrapleural pneumothorax. The region that is freed tends to become adherent against the mediastinum. I do not imagine that it is a complete readherence, so

there is a weakness there. However, in the Semb apicolysis, you have the same weakness, as there is no bone above the first rib, and there cannot be any periosteum there or on the mediastinum. Still, unless we are going to abandon Semb apicolysis for that reason, I do not believe that is a complete contraindication.

As for keeping air in the space early, air does not stay in these spaces very well the first few days. The patients do get emphysema of the chest wall, but, unless the lung has been lacerated, it ought not to be severe. We have had no really serious emphysema. By aspirating only half of the fluid each of the first three days, it, plus what will accumulate overnight, holds the space. After about a week, they hold the air pretty well.

We have had only this one primary infection, and I really believe I may have injured the lung at the time of operation. I have taken three or four cases with very adherent lungs and peeled them away, but this man's lung would not peel, so after about an hour I became disgusted and quit. I may have done the harm by that time.

REGENERATION OF TRACHEAL AND BRONCHIAL EPITHELIUM

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BRONCHIOGENIC carcinoma is demanding more and more attention from the physician, surgeon, and pathologist. This is evidenced by the ever-increasing literature upon the subject. Noticeably lacking from this literature, however, are descriptions which are concerned with the behavior of bronchial epithelium under adverse circumstances, in spite of the fact that a thorough understanding of the basic histology and physiology of such epithelium, in normal and abnormal environments, is fundamental in any approach to so extensive a problem. This paper is restricted therefore to a report of certain basic characteristics of bronchial epithelium which are concerned primarily with its regenerative properties in both normal and vitamin A deficient animals.

NORMAL HISTOLOGY OF TRACHEAL AND BRONCHIAL EPITHELIUM

It is difficult to obtain microscopic sections of the tracheal and bronchial tree which are cut in exactly transverse planes. An oblique cutting tends to present parts of several horizontal planes with a consequent heaping up of the epithelial cells. This obliquity of sectioning has given rise to such terms as "stratified columnar epithelium," "pseudostratified epithelium," and so forth. However, in sections which are cut in approximately transverse planes, the epithelium is shown to consist of a thin row of flat elongated cells which rest upon the peripheral fibrous strands of the submucosa. These cells which comprise the basal layer are separated from the submucosa by a thin basement membrane, which is traversed by only a few very lightly staining collagenous fibrils.

Intimately connected with and resting upon these basal cells is the thicker layer of tall columnar cells which are generally four to six times as tall as they are wide and contain long narrow centrally placed nuclei. The distal ends of these columnar cells are equipped with a well-formed layer of cilia, the length of which is quite constant. The cytoplasm of these columnar ciliated cells is not homogeneous but consists of a fine framework interspersed with numerous small vesiculations. The cells

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vary somewhat in thickness and in curvature on one or both sides, depending undoubtedly upon the pressure exerted by neighboring cells. Spaced irregularly and appearing as elongated ellipsoidal spaces of varying widths are the goblet cells, aptly named because of their likeness to a glass tumbler. These cells are composed primarily of a large clear center which is usually rounded but which narrows to a variable extent toward the free end. Where the section passes directly through their free ends, the openings on the surface may be seen to vary from a narrow slit to a fairly wide mouth. These free ends, although often very narrow, nevertheless show without any doubt a covering of cilia around the stoma. The nuclei of these goblet cells are characteristic, being much wider and thinner than the nuclei of the columnar cells.

I was unable to demonstrate conclusively the occurrence of small cuboidal or triangular-shaped cells located between the basal cells and under, or in juxtaposition with, the columnar cells. These cells, called "Ersatzzellen" by Kölliker,[1] "Keilzellen" by Drasch,[2] and "intermediate cells" by Tuttle and Womack,[3] are supposed to represent a stage in the development of basal to columnar cells. These structures may be bases of columnar cells which appear in obliquely or tangentially cut sections.

This general histologic picture gradually changes as the bronchial tree is descended. The cartilages become thinner and more irregularly spaced; the submucosa narrows; and the serous glands become fewer and fewer and finally disappear. However, the epithelial cell relationship consisting of the underlying layer of flat basal cells and an overlying layer of ciliated columnar and goblet cells remains quite constant until the terminal bronchioles are reached. In the region of the bronchioles the goblet cells disappear, the columnar cells are flatter, and there is a gradual loss of cilia. Terminally, the respiratory alveoli, as classified by Miller,[4] are covered with only one layer of flattened cuboidal cells, a continuation of the basal layer.

Before the conclusion of this histologic description, one further point should be mentioned. Throughout the early and even the late medical literature, there has been an academic controversy as to the origin and mode of production of the goblet cell. Earlier writers, notably Fleming,[5] believed that the goblet cell arose as a separate entity from the basal cell. Others, such as Knauff,[6] believed that these goblet cells presented only a mucous degeneration of the commoner ciliated columnar cell. In this present study, the ordinary columnar cells were highly vesicular and often were distended with vesiculations or cytoplasmic "clear areas" which varied markedly in size and sometimes coalesced into comparatively large vacuoles. As these vacuoles of mucus collected, the cells enlarged and the nuclei were elevated to a higher level in the cell. It is only a further step for such mucous vacuoles to distend the cell completely, and finally to discharge upon the surface of the epithelium.

These resulting goblet cells always retained their covering of cilia. Such an observation substantiates the belief of Knauff that the goblet cell is but an altered form of the common columnar cell. Furthermore the reversibility of the process may be shown, for the goblet cell after discharging its mucus upon the surface returns to the typical columnar cell for its resting phase.

EXPERIMENTAL PROCEDURES

In order to study the growth or regeneration of this bronchial and tracheal epithelium, experimental animals were used. The white rat (Wistar strain) was the animal of choice for several reasons. From the histologic standpoint, the rat presented fundamentally the same picture as that seen in man; as an experimental animal its reactions were quite constant and dependable; its very abundance implied more specimens; and it was somewhat more sensitive to dietary deficiencies than other species.

The simplest method by which to observe regenerative phenomena is, perhaps, the artificial production of some simple, measurable, acute injury and the observation of the healing process at frequent intervals thereafter. For this purpose the rats that weighed 150 to 200 Gm. were selected. Under ether anesthesia, an 8 mm. incision was made low in the neck; the trachea was isolated and, because of its extremely elastic nature in this animal, was dislocated upward to such an extent that a small tracheotomy could be done just above the bifurcation. Through the opening made by tracheotomy an area of epithelium usually measuring 1.5 to 2.0 mm. in diameter was removed with a modified iridectomy forceps and a small scalpel. It was found that closure of the tracheal opening was not needed, but merely a skin suture to close the incision. Operation was performed on 200 rats in this manner. These animals were killed at intervals of from fifteen minutes to ninety days following this injury. The injured region was excised, fixed, and sectioned serially in most instances. In order to mark accurately the exact site of the injury, a small portion of the smooth muscle bundle running posteriorly between the open ends of the cartilaginous rings was also removed. This constant bundle of muscle did not regenerate, and the area from which it was removed showed fibrosis in microscopic sections and thus effectively marked the injured site for identification.

For the study of this epithelium under markedly abnormal conditions, a group of animals was fed a diet deficient in vitamin A. These animals were killed at various intervals, and the conversion or metaplasia of the normal lower respiratory epithelium into a definite, stratified squamous epithelium was followed. Likewise tracheotomy, with the removal of an area of epithelium, was accomplished in a group of rats in such a state of vitamin A deficiency that the entire respiratory epithelium had become keratinized. In like manner, these animals were killed at corresponding intervals, and the regenerative processes of this abnormal type of epithelium were studied.

In addition, a drug, colchicine hydrochloride, was given to a number of normal and vitamin A deficient animals in sufficient dosage to cause a definite mitotic stasis. Following its introduction by Lits[7] and Dustin,[8] this drug has proved of marked value in demonstrating various types and rates of experimental hyperplasias. The present controversy concerning the exact mode of action of this drug in producing the abnormal metaphasic arrest of mitosis or the controversy as to its possible karyokinetogenic properties does not concern this study. However, the use of such a drug made possible several definite observations which well warranted its administration.

OBSERVATIONS

Regeneration in the Normal Animal.—Examination of sections of tissue removed at intervals of fifteen to thirty minutes after the production of injury showed no more than was to be expected. Hemorrhage into the injured area, dilatation of the surrounding blood vessels, and the formation of fibrin upon the surface were the prominent features. Submucosal edema soon made its appearance, and the infiltration of mononuclear cells into the surrounding areas was apparent. After longer intervals of time, such as four or six hours, there were evidences of beginning organization of the hemorrhagic exudate, and the first signs of epithelial cell regeneration were observed. This regeneration consisted in the formation of flat pavementlike cells overlying the normal basal cells at the extreme periphery of the injured area. A few of these cells were covered with cilia and undoubtedly represented the first attempts of the basal cells to perform their function, that is, the production of ciliated columnar cells. This type of response resulting in the regeneration of a surface layer from incompletely removed basal cells was seen on numerous occasions and especially accentuated in later sections (Fig. 1).

In animals killed eight or ten hours following injury, epithelium was regenerating in a characteristic manner. Spreading from the periphery of the injured area out over the denuded surface, which at this time was essentially an immature type of granulation tissue, the epithelium consisted of multiple layers of flat, deeply staining cells. This process was fundamentally a beginning "squamatization," but nevertheless in it here and there on the surface a poor or feeble attempt at the production of cilia was being made. This indicated that, even if these regenerating cells had changed in their form and manner of growth, they still remained endowed with their former properties to some extent (Fig. 2). At this point it was apparent that, where definite epithelial regeneration took place, it did so only over areas which had been prepared to receive it. In other words regeneration took place only over a suitable (nutritive) scaffold. Variable extents of epithelial regeneration had occurred in all specimens taken twelve hours following injury. The extent of regeneration at any interval following the operation was inversely proportional to the depth of the injury and to the amount of inflammatory reaction which had ensued. These two factors, depth of injury and infection, determined the type and quality of the basal structure over which the epithelium must grow. This point was amply demonstrated in the examination and comparison of sections which were obtained twenty-four hours following injury. In some sections where the submucosa was comparatively free from inflammatory products a flat stratified type of epithelium had completely covered the defect. In such an epithelium the many mitotic figures were conclusive evidence of a

very active type of regeneration. The top layer of these squamatized cells was producing minute masses of cilia, and, in addition, a few of the cells were already vacuolated with clear cytoplasmic inclusions as

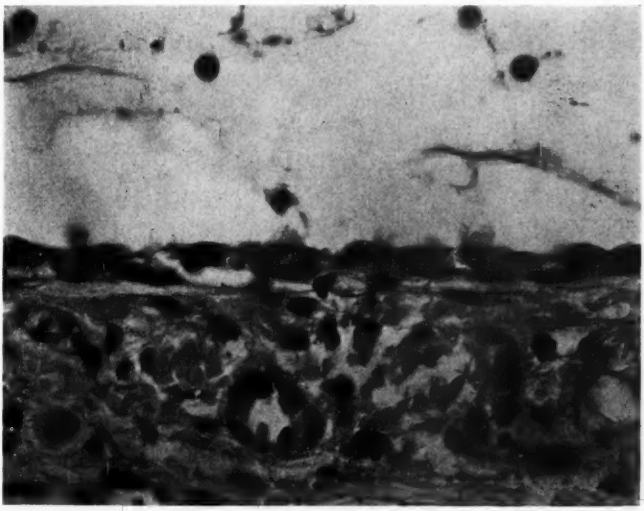

Fig. 1.—Early formation of ciliated columnar cells (×600).

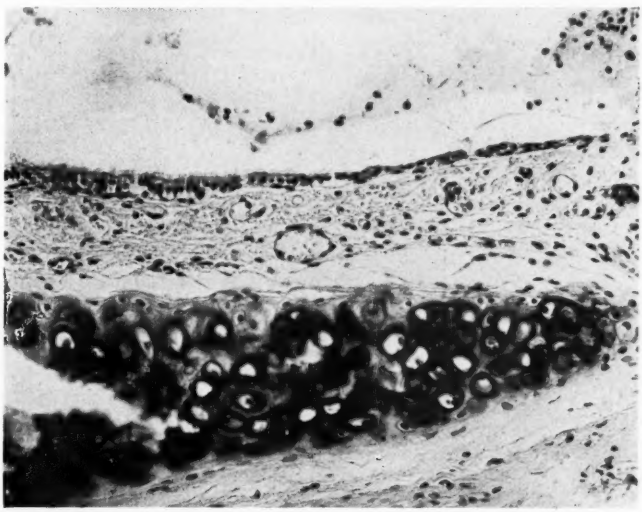

Fig. 2.—Regeneration of basal cells extending from normal epithelium (×85).

if attempts at formation of mucus were being made. This cytologic appearance of rapid regeneration was in marked contrast with that in certain animals, killed at the same intervals, but wherein more severe

inflammatory reactions were present, and in which the epithelial regeneration had progressed to only a slight degree.

However, in most of the animals the entire defect was fully epithelialized by the end of the third day following the original injury. In each instance, this epithelialization was of a squamatized type, that is, composed of multiple layers of flat, irregular, deeply staining cells. Nevertheless, no matter how much like a typical stratified squamous type this regenerative epithelium appeared, formation of keratin was never observed. Keratin, an insoluble sulfur-containing protein, requires a squamous cell of a definite physiochemical make-up for its production.[9] It follows then that this type of cell which formed an epithelium over this injured area was not a metaplastic cell, but merely a more rapidly proliferating cell which retained all of its former fundamental characteristics. This fact, that such epithelium was not basically changed, was brought out in the examination of sections taken from three to ten days following injury. In all of these, as soon as complete epithelialization of the defect had taken place, the epithelium appeared to revert to a more normal type. The success of this reversion seemed to be directly proportional to the quality of the underlying submucosa. If the submucosa remained firmly packed with inflammatory products, swollen with edema, and supplied with small, infrequent blood vessels, then the epithelium covering this area remained thin and consisted of only two or three layers of deeply staining cells with large nuclei. There was no evidence of the formation of multiple layers or of the production of cilia or mucus. On the other hand, where the submucosa contained a loose stroma with well-developed blood vessels and lymph spaces, the overlying epithelium was of the more normal type. The cells of such an epithelium were larger, contained smaller nuclei, and showed to varying extents the formation of cilia and the production of mucus.

The deduction that the epithelial structure in such instances was dependent upon the underlying stroma and that the epithelium played only a passive role was well demonstrated by other findings. Thus, if a microscopic section was made of a region a short distance from the point of direct trauma but through an area where inflammation with marked cellular infiltration prevailed, then the overlying epithelium lost its normal appearance and took on the properties of squamatization (Fig. 3). This process was observed so commonly that the assumption was made that this tracheal and bronchial epithelium rather constantly reverted to a more immature (perhaps more easily sustained) type of epithelium under conditions of excessive distress.

For a study of the final stages of regeneration, a period of three months was allowed to elapse from the time of injury until the animal was killed. One should expect that, after this period, the healing process would be complete. Some sections obtained after this prolonged interval showed, rather surprisingly, an almost complete return to the normal

state. The regenerated epithelium consisted of flat basal cells, topped by cuboidal or columnar cells, the cilia of which in places were irregular but nevertheless present. Goblet cells were also fairly numerous. Such appearances marked this epithelium as essentially normal. In contrast, however, other preparations, after the same time interval, showed that a reversion to the normal state had been accomplished only slightly. In these sections, the submucosa was filled with inflammatory debris—a rather typical picture of chronic inflammation—and the overlying epithelium still remained in an irregularly stratified form. There were no cilia or goblet cells, and, as a whole, such sections were very similar to those obtained at three- or four-day intervals. In spite of this rather prolonged period in which the epithelium was subjected to such conditions, commonly described as chronic irritation, there were no proliferation, no well-marked epithelial pegs reaching down into the submucosa, and no excessive heaping up of the surface layers.

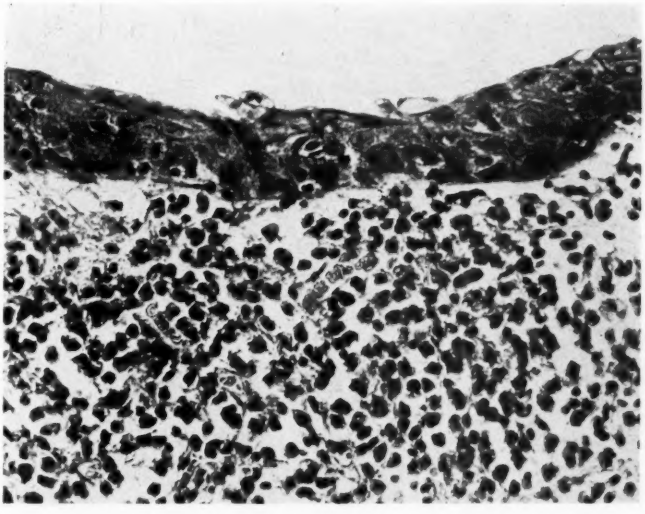

Fig. 3.—Bronchial epithelium overlying an acutely inflamed submucosa (×400).

In review, tracheal and bronchial epithelium, highly specialized though it may be, is capable nevertheless of a comparatively rapid regeneration; initially, this regeneration proceeds as a pseudostratified variety; and subsequently at variable later stages regeneration results in partial or complete return to the normal pre-existing structure.

The Use of Colchicine.—In a number of animals colchicine hydrochloride was used as an aid in this study. Principally because of its effect upon mitosis, it presents to the investigator evidences of cell regeneration which otherwise would be most difficult to demonstrate. After a preliminary investigation to determine the optimal dosage and the

optimal time interval during which the drug was allowed to act, it was found that 0.1 mg. per 100 Gm. of body weight of the animal, given subcutaneously as a 1 per cent solution in physiologic saline, eight hours previous to death would result in the maximal effect of the drug.

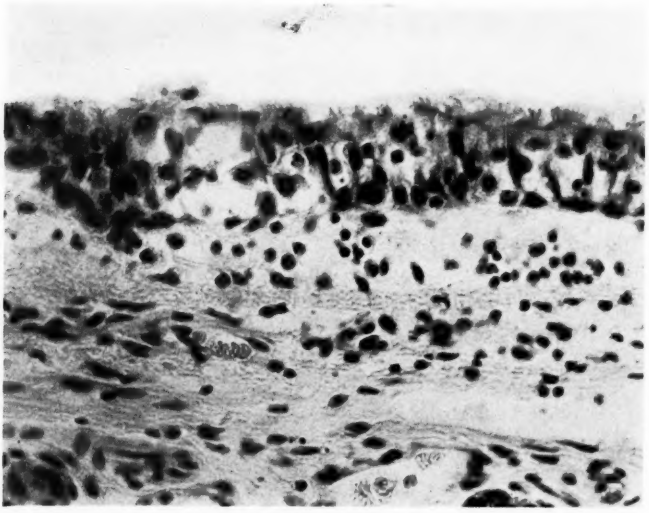

Fig. 4.—Mitotic figures within the ciliated columnar cells (×400).

Following administration of colchicine in this dosage to normal animals microscopic sections of the trachea and bronchi showed but a few scattered mitotic figures, and most of these were found only in the basal cell layer. The paucity of mitoses in this type of epithelium was very apparent when contrasted with sections of the normal esophagi of the same animals in which many mitotic figures occurred, often as many as thirty or more per high-power field. In considering this fact that, even with the use of colchicine, mitoses were infrequently encountered, it became apparent why most investigators have rarely seen cell division in the normal trachea and bronchi. It would follow then that normally respiratory epithelium is a very stable structure; that is, it does not lose its various epithelial elements rapidly and thus is not in the normal state undergoing a constantly active type of regeneration.

However, in those animals in which tracheotomy was done, followed by excision of a limited area of epithelium, the use of colchicine brought into view many mitoses. In such instances as these, a strong stimulus to regeneration was being applied, and colchicine merely acted as a static agent to hold such mitoses in the metaphase for a given period (Fig. 4). These figures were predominantly found in the basal or germinative layer where the most active regeneration was taking place. However, many mitotic figures were also found in the columnar and

goblet cells. Indeed, in one section examined, eighty of a total of 250 mitotic figures counted were within the columnar cells. On the assumption made by many other investigators that with the use of colchicine a rough estimate can be made of cell hyperplasia or rather of the tempo of regeneration, it will be seen that, when placed under an irritative or stimulative influence, respiratory epithelium is capable of very rapid regeneration.

Further examination of sections from these colchicinized animals demonstrated also that the mitotic figures occurred most frequently within the epithelium immediately around the injured area just distal to the actual "leading" or "growing" edge. In other words, the most active regeneration was found a short distance from the denuded surface where the epithelium had become more firmly established and where proper nutritive conditions prevailed. It was in this location that the epithelium was beginning to thicken and take on a more normal appearance. This point has a similar corollary in the healing of gastric lesions, for here the most active regeneration takes place at the periphery of the crater of a gastric ulcer and not at the extreme epithelial edge.[10]

A third point worthy of mention was also well demonstrated by the use of colchicine. In areas where there was marked submucosal inflammation, the overlying epithelium showed comparatively few mitotic figures, whereas, in the same section, mitotic figures were in greater abundance within the epithelium covering a noninflammatory area of submucosa. This observation again merely demonstrated the fact that the behavior of such an epithelium is directly dependent upon the state of the underlying tissues.

The Behavior of Respiratory Epithelium in the Vitamin A Deficient Animal.—Although manifestations within the epithelium of the tracheobronchial tree resulting from diets deficient in vitamin A had been observed originally by Osborne and Mendel,[11] and McCollum and Davis,[12] it was left to Wolbach and Howe[13] to describe such changes completely and accurately. These investigators demonstrated the replacement of normal cuboidal or columnar epithelium of the respiratory tract from the nares to the bronchi by a keratinizing stratified squamous epithelium in vitamin A deficient guinea pigs. It was shown that this epithelial transition took place primarily by the formation of new cells beneath the original epithelium, that this formation was essentially a multiplication of the basal layer, and that this type of growth proceeded at such a rapid rate that numerous layers (stratification) became apparent. Wolbach and Howe further showed that the overlying columnar layer, although present for some time, finally could not withstand the change and was replaced by a thin pavement type of epithelium. When this process was allowed to persist, these authors found that this rapid proliferation continued with the sloughing off of large amounts of this

keratinized epithelium. Further, occlusion of the various bronchi by this discarded material occurred, and secondary changes due to inflammation, atelectasis, bronchopneumonia, lung abscess, and so forth were the result. These observations have been duplicated by many workers, but it was pertinent to this present study to measure and contrast roughly the regenerative properties of this markedly altered, metaplastic type of epithelium with that found in the normal animal.

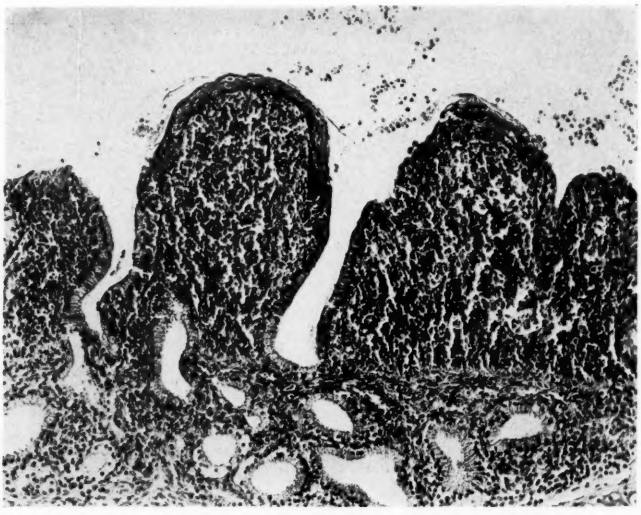

Fig. 5.—Inflammatory polyps occurring within the trachea of a vitamin A deficient rat (×120).

For this purpose fifty young rats, the day after weaning, were placed upon a commonly used vitamin A deficient diet, and at frequent intervals the progress of the metaplastic change was observed histologically as it occurred within the bronchial tree. Once established, this keratinizing squamous epithelium showed marked evidences of rapid proliferation. Epithelial pegs dipping deeply into the submucosa were common. Frequently, polyps of the inflammatory type were present even to the extent of general polyposis of the entire trachea (Fig. 5). Further confirmation of the extreme rapidity with which this epithelium was growing was found by the administration of colchicine to such vitamin deficient animals. In these instances, sections of the trachea and bronchi revealed often as high as 60 per cent of the epithelial cells in the state of mitosis.

The ease with which vitamin A, whatever its physiologic mechanism, would exert its effect upon the metaplastic epithelium was striking. Once fully established throughout the entire respiratory tract in the deficient animal, this squamous epithelium would revert fairly rapidly,

that is, within fourteen days, to the normal columnar ciliated epithelium after the exhibition of massive daily doses of this vitamin. Such a reversibility of cell type has been shown previously on many occasions by other investigators. Of interest to our studies, however, was the type of response that this artificially created epithelium would make in the face of injury. To observe such a response and to compare it with that which was already noted in the normal animal, several of these vitamin deficient rats were subjected to tracheotomy, in the manner described previously, and a section of tracheal epithelium was removed.

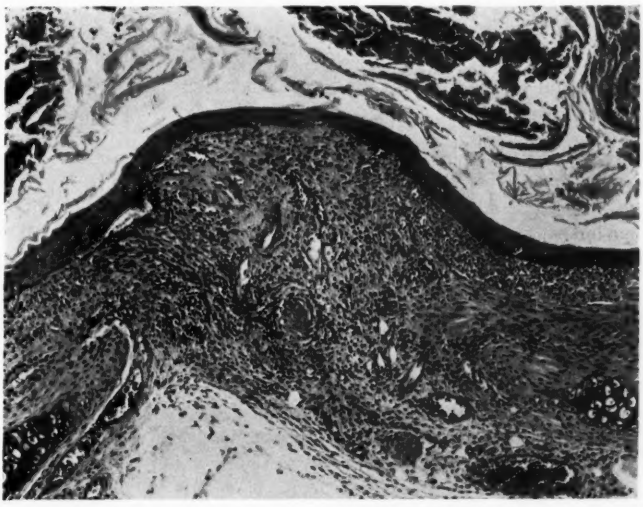

Fig. 6.—Complete epithelialization in a vitamin A deficient animal thirty hours following injury (×85).

At stated intervals, separate animals were killed and the regenerative processes noted. Histologic examination showed that epithelialization of this defect occurred by the production of a fairly thick layer of keratinizing squamous epithelium. Rather surprising, however, was the fact that this type of epithelial covering was thrown over the denuded surface, in each instance, in less than thirty-six hours following the operation (Fig. 6). This observation again attested the extreme rapidity of growth that this epithelium manifested during periods of stress. Although measurements at various intervals were not necessary, it was apparent that healing progressed at least twice or three times as rapidly as was previously found in the normal, nondeficient animal.

COMMENT

From these studies it may be generally summarized that (1) following a severe injury there is a rapid regeneration of tracheal epithelium to cover the denuded surface; (2) the rapidity of such regeneration is

dependent upon the quality of the stroma across which the epithelium must grow; (3) the healing process is essentially a function of the basal cells which form the initial layer; (4) the form of the subsequent regeneration resulting in secondary layers of cells is again dependent upon the quality of the submucosa; (5) the constantly found squamatized type of epithelium is not a true stratified epithelium but merely an altered form of the original; and (6) there is in all these regenerations a constant tendency to reform a normal epithelium. These points have been made in order to show that this regeneration does not involve metaplasia. "Metaplasia," as Virchow first intended the meaning, implies a complete change not only in cell structure but in cell function, a change which in most instances is permanent and which results in the formation of an entirely different type of tissue.

For this present instance, the word "protoplasia," as used by Fried,[14] is much more apt, for "protoplasia" implies only variation in structure and function of a more or less pleuripotent cell. Thus, when one is speaking of "squamous epithelium" of this type, there should be some qualification as to whether it is a "true" or a "pseudo" type. A type of pseudosquamous epithelium, not involving keratin formation or the formation of epithelial pearls or any other characteristics of true squamous epithelium, has appeared in numerous instances in human lungs in cases of tuberculosis, postinfluenzal bronchitis, whooping cough, asthma, bronchiectasis, and so forth. There has not been a proved etiologic relationship between any of these conditions and bronchiogenic squamous cell carcinoma. Further, a squamous carcinoma of the lung, showing keratin formation, and so forth, such as is sometimes encountered, depends perhaps upon a more radical change in epithelial cell type than has been seen in any of the studies described in this paper.

Concerning the use of colchicine, it should be commented that the mitoses which appeared in such abundance were not normal figures and considerable speculation now exists as to the method by which such mitoses are produced. Nevertheless, the present consensus, in which the writer concurs, prevails that colchicine mitoses appear in proportion to the amount of hyperplasia in any given tissue at any given time and colchicine does not produce mitotic figures within cells which normally are incapable of self-reproduction.

Fried,[15] expressing a rather widely held opinion, has stated with some emphasis that, because goblet and columnar cells represent a completely differentiated type of cell incapable of any regeneration or reproduction, they are, therefore, impotent to form cancer. In the case of tracheal and bronchial glands he also used this reasoning. However, in this present instance regeneration or cell division was shown to take place frequently not only within the columnar and goblet cells of the trachea and bronchi, but within the columnar, mucus-secreting cells of their accompanying serous glands when such structures were stimulated prop-

erly to increased growth. It should not be construed, of course, that, because reproduction of such cells has been shown, a direct origin for a malignant lesion may be found in these cells.

In addition, the use of colchicine in this study also made possible the observation that in a normal state the lower respiratory epithelium is not regenerating actively and remains in a "resting stage" a good share of the time. However, should a stimulus for growth be applied, this normally quiet epithelium is capable of flaring up into a very active one which at once presents marked proliferative properties.

With the use of a vitamin A deficient diet this same property was further demonstrated, for in this instance tracheal and bronchial epithelium, when properly stimulated by the addition or subtraction of certain definite factors necessary for normal cell behavior, quickly changed its whole structure and became a rapidly proliferating one. Conclusions should not be drawn from such an observation, but it is of interest to speculate that the extreme lability of such a mucosa might be one factor in the frequency with which bronchiogenic carcinomatous growths are found.

SUMMARY AND CONCLUSIONS

By the method of direct injury, regeneration of the epithelium of the trachea has been studied in the normal and in the vitamin A deficient animal. From such studies the following facts have been observed.

1. Tracheal epithelium consists essentially of a well-defined basal layer of cells with an overlying layer of ciliated columnar and goblet cells, the latter being a variant of the former.

2. Normally this epithelium is slowly regenerative.

3. Under conditions of stress, this epithelium may alter its form and show rapid regenerative properties.

4. Once altered in form, the epithelium always attempts to return to a normal state.

5. The success of this attempted return to a normal state is directly proportional to the quality of the underlying submucosa.

6. A colchicine technique will demonstrate the rapidity and locations of the greatest regenerations.

7. Fully differentiated columnar and goblet cells are capable of reproduction.

8. Metaplasia occurs during states of vitamin A deficiency.

9. This metaplastic type of epithelium has the power of extremely rapid proliferation.

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THE TECHNIQUE OF THE LIGATION OF THE PATENT DUCTUS ARTERIOSUS

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SURGICAL ligation of the patent ductus arteriosus was first suggested by Monroe[1] in 1907 before the Boston Surgical Society. He had seen a baby die of cardiac failure with no other cardiac lesion than a patent ductus arteriosus. He demonstrated at the autopsy table that it would be technically possible to ligate this structure. Moreover, he suggested that "the physiologists' apparatus for maintaining artificial respiration" should be used if necessary. Following Monroe's suggestions some twenty years passed before intratracheal anesthesia was widely used clinically, and it was thirty-one years before a patent ductus arteriosus was successfully ligated.

Although O'Shaughnessy[2] and Strieder[3] had previously operated with the intention of ligating a patent ductus arteriosus, Gross[4] did the first successful ligation on Aug. 26, 1938. Since that time Gross[7, 8] and others[5, 6, 9-11] have operated upon an increasing number of patients. We wish to record seven operations performed at the Hospital of the University of Pennsylvania.

CASE REPORTS

CASE 1.—L. G. was a girl, aged 16 years. The murmur was discovered at birth, and the patient had been followed in our cardiac clinic for about five years. She was developing increasing cardiac enlargement and had dyspnea on slight exertion. The ductus was ligated on July 19, 1940. She has had an excellent result. The heart has decreased 20 per cent in size and the patient no longer has dyspnea on exertion.

CASE 2.—V. T., a girl aged 5 years, was always underdeveloped. The cardiac murmur was discovered recently. The heart had not yet enlarged, but the patient's pulse rate increased more than normal on exertion. After successful ligation of the patent ductus arteriosus on Aug. 2, 1940, the child has shown considerable general improvement. The heart size has decreased approximately 15 per cent.

CASE 3.—S. B., a girl, aged 18 years, had a large heart and was quite dyspneic on slight exertion. At operation on Oct. 6, 1940 the ductus was found to be very thin and was inadvertently torn. The subsequent hemostasis was improperly secured, and the patient died thirty-six hours later. (See discussion.)

CASE 4.—A. B., a boy, aged 21 years, was underdeveloped for his age. He had had chills and fever for three months, and the blood culture showed 500 colonies of

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Streptococcus viridans per cubic centimeter of blood. At operation on Nov. 18, 1940 the ductus was found to be unusually thin. It apparently was not the seat of a vegetative process as there was no edema. The ductus was ligated by the technique shown in Fig. 4 except that only one ligature was used (the one at the aortic end of the ductus). The thrill and murmur changed in quality but were still present. The course of the endocarditis apparently was not affected by the operation. The patient died seven months later. At autopsy the ductus was found to be completely closed. There was an intraventricular septal defect. The pulmonary valves were partially destroyed. The vegetations were on both sides of the heart, but chiefly on the right. There were no vegetations in the region of the previously patent ductus arteriosus. These pathologic findings will be reported in greater detail in a later communication.

CASE 5.—J. M., a boy, aged 4 years, was considerably underdeveloped physically and mentally. He had not yet learned to talk. His heart had not enlarged, and there were no signs of cardiac failure. At operation on March 28, 1941, the ductus arteriosus was easily ligated. The murmur disappeared. The cardiac size was not materially changed after operation.

CASE 6.—P. C., a girl, aged 12 years, was underdeveloped for her age. She had slight cardiac enlargement. There were no signs of cardiac decompensation. She repeatedly had R.B.C. in her urine with an increased Addis count. She continually had congestion of her upper respiratory tract which was thought to be largely on an allergic basis. After considerable study the patient was operated upon on June 22, 1941. The ductus was short but not unusually thin so it was easily ligated by the technique shown in Fig. 3. The patient left the hospital in good condition.

CASE 7.*—L. A., a colored woman, aged 35 years, was considerably underdeveloped. She had had "heart trouble" for seven years. Her heart was enlarged. She had been in moderately severe cardiac decompensation off and on for a year. She had fever up to 100° to 102° F. daily with occasional remissions for about three months. It was our belief that she already had subacute bacterial endocarditis or endarteritis. However, six blood cultures showed no growth. At operation on June 27, 1941 a short ductus which was not unusually thin was ligated by the technique shown in Fig. 3. There was no acute edema in the region of the ductus. The murmur disappeared entirely. The temperature returned to normal, and the patient left the surgical ward in good condition.

In a later communication these cases will be presented in greater detail with indications for operation, and results from the standpoint of the cardiologist. In general, we feel that, if a large ductus arteriosus persists after 5 years of age, ligation is justifiable since it has been shown that many such patients develop some type of trouble at a later date.

OPERATIVE TECHNIQUE

The operative technique as described by Gross was found to be very satisfactory in our first two cases. In each of these patients the ductus arteriosus had a wall about as thick as that of the pulmonary artery. Under these circumstances it is a relatively simple matter to free the ductus from the surrounding structures so that ligation can be performed successfully. However, in our third patient we

*Case 7 was operated upon at the Philadelphia General Hospital on the service of Dr. J. P. North.

found a very thin-walled ductus. It was freed without difficulty on three sides, but in the attempt to free the posterior aspect the ductus was torn. The hemorrhage was not properly controlled, and the patient died thirty-six hours later. Gross,[8] Jones,[5] Humphries,[11] Touroff[9] and Miangolarra[10] have had the misfortune of tearing the ductus when it was very thin-walled or friable due to vegetative endarteritis. Fortunately, the subsequent hemorrhage has often been controlled without a fatality.

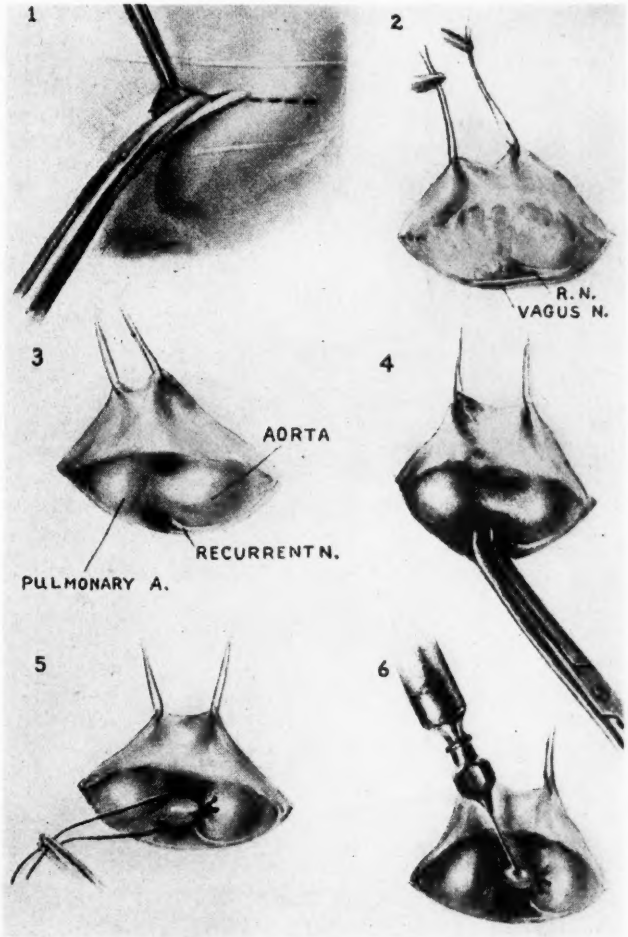

Fig. 1.—Illustration taken from Gross and Hubbard.[8]

However, when the operative technique outlined by Gross is used, the likelihood of tearing the ductus is definitely increased if it is very thin-walled. By his technique, a curved hemostat is gently worked around the ductus in order to separate it from the adjacent structures (Fig. 1). It probably has been this maneuver which has resulted most frequently in tearing the ductus.

SUGGESTED VARIATION IN TECHNIQUE

Tearing of the ductus can be avoided on the anterior surface where it is clearly visualized, but trouble may be encountered when blind dissection is attempted posteriorly. It is suggested, therefore, that all contact with the posterior wall of the

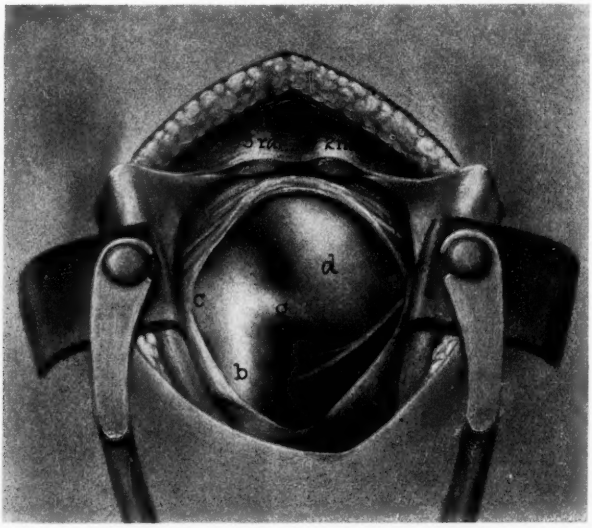

Fig. 2.—The usual incision in the left second interspace for the exposure of the patent ductus arteriosus. *a*, Right pulmonary artery; *b*, left pulmonary artery; *c*, pulmonary artery; *d*, arch of aorta; *e*, phrenic nerve; *f*, left recurrent laryngeal nerve; *g*, patent ductus arteriosus.

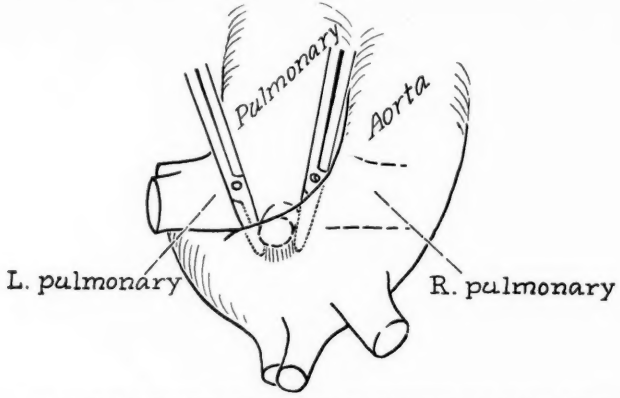

Fig. 3.—A method of avoiding contact with the posterior wall of the ductus arteriosus when there is sufficient areolar tissue between it and the left main stem bronchus. The dissection is carried well posterior to the ductus wall on either side of the ductus before an effort is made to connect these spaces.

ductus arteriosus be avoided. This may be done (Fig. 3) by using a straight hemostat for the blunt dissection between the aorta and the pulmonary artery. In this manner a space may be opened between these two great vessels on either

side of the ductus arteriosus, extending well behind the posterior wall of the ductus. These spaces may then be connected posteriorly with a curved hemostat which at all times is kept well away from the posterior wall of the ductus. This technique was used in Cases 5, 6, and 7 and may be employed when there is sufficient areolar tissue between the posterior wall of the ductus and the underlying left main stem bronchus.

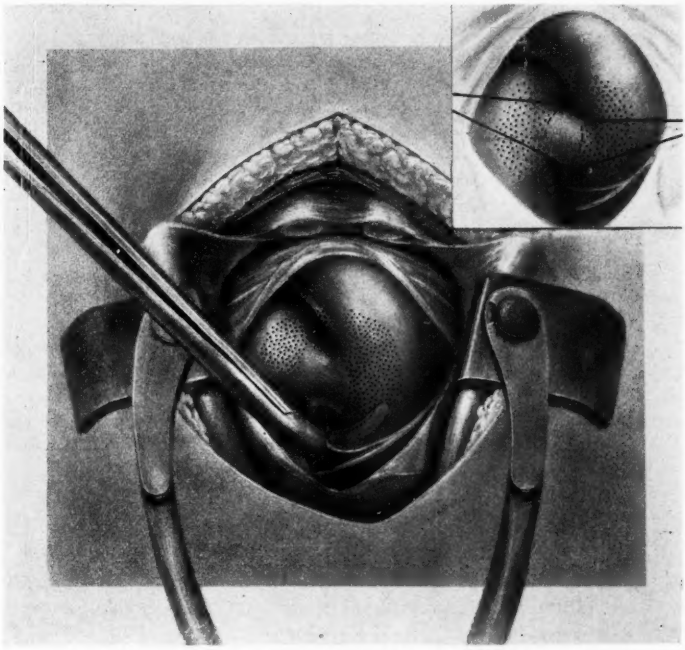

Fig. 4.—A method of ligating a thin-walled ductus arteriosus when there is insufficient areolar tissue between it and the left main stem bronchus to allow the technique shown in Fig. 3. The ductus is not freed posteriorly so that there is no contact with the posterior ductus wall.

TECHNIQUE SUGGESTED FOR HAZARDOUS CASES

If there be very little areolar tissue behind the ductus and great vessels, the posterior blunt dissection may of necessity come close to the posterior wall of the ductus arteriosus. If such be the case, or the ductus be unusually thin-walled, or edema indicates the presence of endarteritis, it is suggested that the blunt dissection posterior to the ductus arteriosus be abandoned. In its place the dissection may be carried out against the relatively thick-walled pulmonary artery and against the even thicker-walled aorta, the posterior wall of the ductus being left undisturbed (Fig. 4). In order that this procedure may be carried out, there must be a more extensive exposure of the mediastinal structures. This can be obtained by increasing the opening in the mediastinal pleura. The aorta should first be exposed anteriorly, well to either side of the ductus. By pushing the recurrent laryngeal nerve aside, the finger or an instrument can find a plane around to the posterior aspect of the aorta. This dissection is simple since one can work against the thick-walled aorta. The posterior surface of the aorta can thus be exposed proximal

and distal to the ductus arteriosus. One finger is placed behind the aorta distal to the ductus and curved to a point proximal to the ductus behind the aorta. By staying close to the wall of the aorta a plane of cleavage may be found around the inferior surface of the aortic arch proximal to the ductus, between the aorta and the right pulmonary artery. A ligature can then be placed around the aortic end of the ductus arteriosus although the posterior wall of the thin-walled ductus has not been separated from the adjacent structures. This technique was used in Case 4.

This same procedure can then be carried out on the pulmonary artery. The left pulmonary artery should be exposed in the same manner as if one were going to do a pneumonectomy. The posterior wall can then be freed by blunt dissection with the finger. The finger can be carried across the midline so that the right pulmonary artery is in part freed posteriorly. A plane of cleavage can then be established around the superior surface of the right pulmonary artery, and a ligature placed around the pulmonary end of the patent ductus arteriosus in the same manner as was previously done at the aortic end. The posterior wall of the thin-walled ductus is not freed and is left adherent to the neighboring posterior structures. Heavy silk ligatures are used to prevent any possible cutting through of the vessel wall. The patent ductus may thus be completely closed by two widely placed ligatures (Fig. 4).

This procedure obviously requires considerably more blunt dissection in the mediastinum than the simple technique proposed by Gross. It is not suggested that this modified procedure be used in all cases, but only in those instances in which a very thin-walled ductus creates the hazard of injury to the ductus wall, in those cases with subacute bacterial endarteritis with implantation in the ductus, or when insufficient areolar tissue exists between the ductus and the left main stem bronchus to allow the technique described above (Fig. 3).

DISCUSSION

We do not believe that the ductus would have been torn in Case 3 if the technique outlined above had been used. Moreover, it is felt that a fatality could have been avoided if a different procedure had been used after the ductus was torn. Our error was in attacking the bleeding point directly, attempting first to pass ligatures around the ductus on either side of the tear, and, when this failed, to clamp both ends of the ductus with large hemostats. During these procedures, due to a bloody field and inadequate exposure, the patient was almost exsanguinated before the hemorrhage was controlled. In desperation the left pulmonary artery was finally ligated with the ductus arteriosus to stop the bleeding. This was felt to be a justifiable procedure because of the experience of Rienhoff[12] in ligating one pulmonary artery as the first stage of a two-stage pneumonectomy. The patient died thirty-six hours later without further hemorrhage. An autopsy could not be obtained so that it is not known whether damage to the left lung was a factor in her death.

SUGGESTED MANAGEMENT OF A TORN DUCTUS ARTERIOSUS

The following technique has been worked out as the result of our fatality in Case 3.

It is suggested that, if a ductus arteriosus be torn, the bleeding be controlled temporarily by packing, and steps be taken to prevent a

serious hemorrhage during the subsequent dissection. The pericardial sac should be opened, and a piece of hernia tape placed loosely around the pulmonary artery in the same manner as for a pulmonary embolectomy (Fig. 5). If bleeding should be profuse at any time, it can be controlled momentarily by compressing the pulmonary artery. One should then proceed to get a wide exposure of all of the surrounding

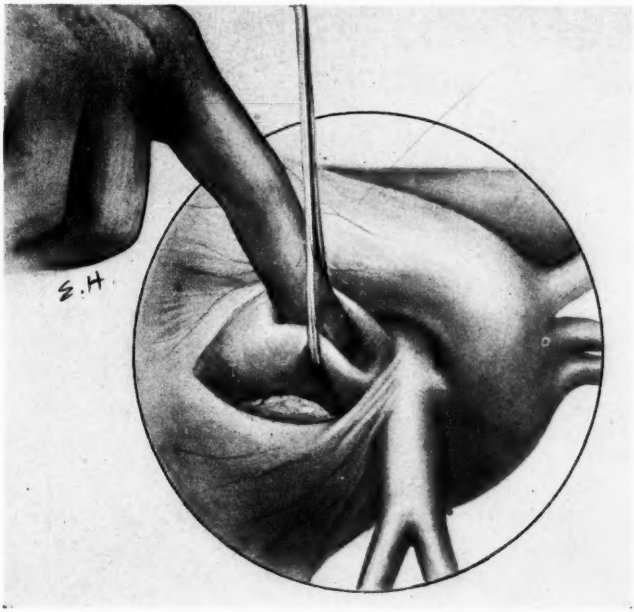

Fig. 5.—If the patent ductus is torn, the pericardial sac may be opened and a hernia tape placed around the pulmonary artery to stop bleeding momentarily in the event of a threatened exsanguinating hemorrhage.

structures. If the ductus had been torn in the middle, it would then be possible to leave the remainder of the posterior wall undisturbed and place two ligatures around the ends of the ductus by the method described above (Fig. 4). If it became apparent that this was impossible, then the ductus should be cut completely across so that the two ends could be closed separately. It is imperative before doing this that adequate exposure be obtained. If the bleeding has not been controlled at the aortic end by a hemostat, an assistant may place his finger over the hole in the aorta while attention is turned to the pulmonary end of the ductus. The operator's right index finger is placed over this opening if it is not already satisfactorily controlled with a clamp. The entire pulmonary artery with both branches should be exposed. The operator's left index finger is then placed around behind the left pulmonary artery so that the bleeding can be controlled from the pulmonary end of the

ductus by closing with the index finger and thumb. The blood is thus allowed to go through the right pulmonary artery only (Fig. 6). The opening in the pulmonary artery may then be sutured with interrupted

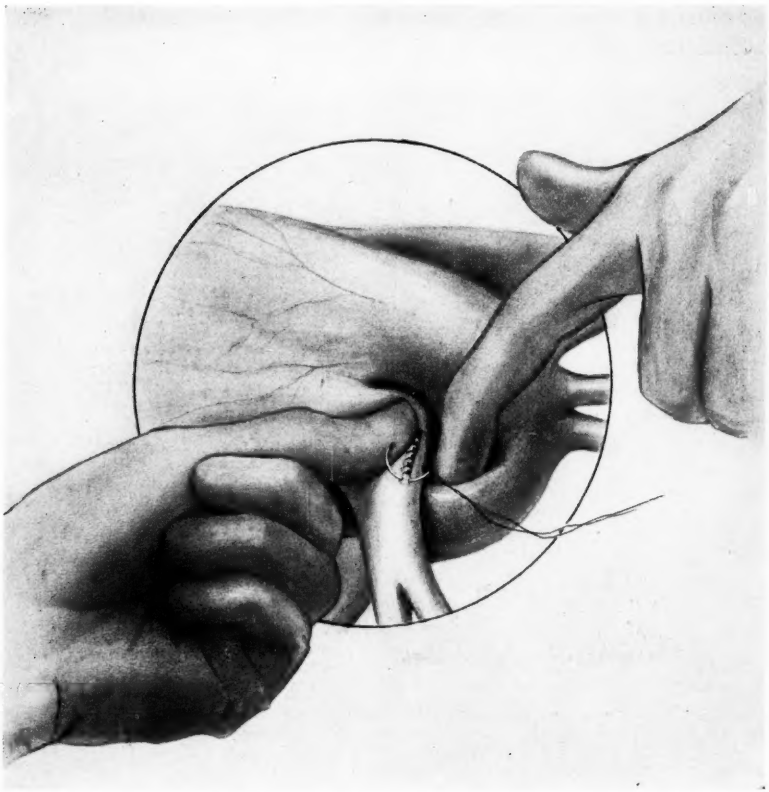

Fig. 6.—If the ductus has to be completely divided, the opening in the aorta may be closed by an assistant's finger. The left pulmonary artery may be compressed between the thumb and forefinger so as to allow the circulation to continue through the right pulmonary artery, while the pulmonary end of the ductus is sutured.

arterial sutures in an unhurried manner. Attention should then be turned to the aortic end of the torn ductus arteriosus.* A piece of

*During the course of the operation in Case 3 the question arose as to whether a clamp could be left on the opening in the side of the aorta with hope of satisfactory healing. It is well known that the urologists frequently leave clamps on the kidney pedicle with satisfactory results. This problem has therefore been investigated in dogs. It was found that when a curved clamp was left on a hole in the side of the aorta, the animal invariably died of hemorrhage. The portion of the wall caught between the clamp stayed there and became necrotic. Rupture occurred at the junction of the normal wall and the necrotic part caught by the clamp. This occurred within four to seven days. When the clamp was removed before this time, fatal hemorrhage likewise occurred. On the other hand, when a clamp was placed on the renal artery it could be removed safely on the fourth or fifth day. Satisfactory hemostasis was obtained when as little as 1 mm. of renal artery remained between the clamp and the aorta. It seems likely, however, that the length of the branch artery which would have to be left between the clamp and the aorta would be greater if one were dealing with larger vessels in larger animals.

hernia tape should be placed around the aorta between the left carotid artery and the left subclavian artery. Compression at this point by the assistant against his finger allows the circulation to continue to the brain while sutures are placed in the aortic wall. This opening should be closed with closely placed interrupted fine silk sutures (Fig. 7). In the event that there should be troublesome bleeding from the needle holes in the aorta, a small piece of muscle should be tied down with a suture and held in close contact by means of a piece of fascia lata brought completely around the aorta and sutured in place to reinforce the sutured area in the aortic wall.

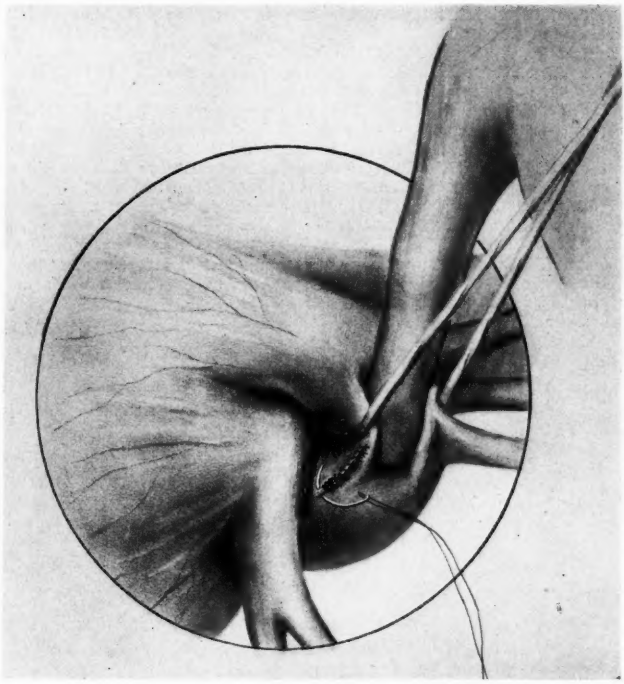

Fig. 7.—When the aortic end of the ductus is being sutured, hemorrhage may be controlled by pressure with a hernia tape around the aorta between the left carotid and the left subclavian arteries. The circulation to the brain is not interrupted.

SUMMARY

1. Seven cases of ligation of the patent ductus arteriosus are presented. There was one fatality due to improper hemostasis following the tearing of a thin-walled ductus arteriosus. One patient with subacute bacterial endocarditis died seven months later of the endocarditis. The other five patients have had excellent results to date.

2. A variation in Gross' technique is suggested to be used under ordinary circumstances.

3. A second variation in technique is suggested to be used in the event that the ligation of the ductus appears to be unusually hazardous.

4. A method of procedure is suggested in the event that a thin-walled ductus arteriosus be torn.

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